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ARTICLE I.

THE ANNUAL DISCOURSE.

# THE EXPANSION OF MEDICINE.

By EDWARD H. BRADFORD, M.D.
OF BOSTON.

DELIVERED JUNE 14, 1899.

NOTE.—At an Adjourned Meeting of The Massachusetts Medical Society, held Oct. 3, 1860, it was

*Resolved*, "That The Massachusetts Medical Society hereby declares that it does not consider itself as having endorsed or censured the opinions in former published Annual Discourses, nor will it hold itself responsible for any opinions or sentiments advanced in any future similar discourses."

*Resolved*, "That the Committee on Publications be directed to print a statement to that effect at the commencement of each Annual Discourse which may hereafter be published."

# THE EXPANSION OF MEDICINE.

Mr. President and Fellows
of The Massachusetts Medical Society:

When that most intelligent observer of human affairs, Benjamin Disraeli, greeted a deputation of medical men with the clever paraphase, "Sanitas sanitatum omnia sanitas," he was, perhaps, wiser than he thought.

The growth of the field of medicine has been great and is still increasing. From the fetich man, the priests at the temple, the writers of medical aphorisms and dogma there has developed in the course of the centuries a profession of practitioners of medicine and the beginning of a science. This expansion is certain to continue with the growth of knowledge. Perhaps the court in the next century, whose decree "Thou shalt not" will be obeyed without compulsion, is that which transcribes with accepted authority the immutable laws of disease.

Is the profession prepared for added responsibilities? An examination of the past record is pertinent in attempting an answer to this query.

The practice of medicine even in the early days of the American Colonies seems to have attracted

to it men of capability. The settlers brought none of the traditions of feudalism, which made the leech and barber surgeon a servant of the chief. The profession was early regarded as a desirable one. In the Dutch Colony of New Amsterdam, the doctor was an official, and in New England almost as much respect was paid to the physician as to the clergyman. Distinction was granted to those who were regarded as educated men. The doctor trained his son to follow in his footsteps and educated him for the purpose as thoroughly as was possible. From the year 1631 to 1805 there always was a Dr. John Clark in Boston for the citizens of Boston to consult. The title was almost hereditary, and there are physicians in the city of Boston to-day whose direct ancestors were practising here a hundred years ago (Warren, Hayward, Bigelow, Shattuck, Homans, Townsend, Cheever, Jeffries, Channing, Reynolds).

Dr. Holmes writes ("The Medical Profession in Massachusetts"): "Many names in the catalogue of the early physicians have been associated in the late period with the practice of the profession, among them, Boylston, Clark, Danforth, Homans, Jeffries, Kittredge, Oliver, Peaslee, Randall, Shattuck, Thacher, Wellington, Williams, Woodward."

The public accorded prominence to the physician. The Medical-Chirurgical Faculty of Maryland a hundred years ago proudly claimed as its motto the line of Homer:

Ἰητρος γὰρ ἀνηρ
πολλῶν ἀντάξιος ἄλλων.

In return, character, broad education, force and high purpose were demanded of the leaders of the profession. Nathan Smith, Gerhard, Elisha Bartlett, Warren, Bigelow, Alonzo Clark, Ware, Austin Flint, Bowditch, and their peers maintained the standard and passed their example to posterity. There has been no lack of achievement from a want in the character of the medical profession.

The education of medical men in America was dependent upon the conditions of American life. In the early provincial days and in a large country with a sparse population with imperfect means of travel, students were, following the English custom, apprenticed to the prominent physician of the neighboring town. They read medicine in the physician's office and performed subordinate work. They lacked schooling, but they had the best of training in graded and increasing experience under the eyes of exacting criticism. As the communities grew, the number of these students increased and the physicians could neither find room nor time for their classes. Schools were formed by the association of several physicians and their following. Energetic and ambitious physicians called themselves professors or induced a struggling neighboring college, ambitious to obtain the distinction of a university with many faculties, to appoint them to chairs. The prominent positions were assigned to the most influential physicians; the younger men hoping for preferment trained themselves by lecturing on the less popular

subjects. Nathan Smith, who founded three medical colleges, was professor of several faculties in each, not finding sufficiently worthy colleagues.

The public was not aware that there was need of rich endowments of medical schools, as the best of practitioners had been bred without laboratories or perfected schools, and as a result, few of our medical schools have endowments for scientific work; consequently a large number of students were and are in many localities obliged to attend foreign schools, if desirous of learning the medical sciences. "Pertransiunt multi et crescit scientia."

The condition of our leading medical school thirty years ago is well described by a recent writer in a statement which older physicians know is not exaggerated.

"In 1869 President Eliot found the (Harvard) Medical School little more than an irresponsible commercial venture. There were no requirements for admission; attendance was required for two courses of lectures only, brief in themselves, and still further abbreviated by the failure of the great majority of students to attend during the summer term. A student who passed successfully five out of nine oral examinations of five minutes' duration each, received a diploma, although he might not know the limit of safety in the administration of morphine, and one had actually killed two early patients in consequence."

As the President said, "Under this system young men might receive the degree of Doctor of Medicine who had had no academic train-

ing whatever, and who were ignorant of four out of nine fundamental subjects." *

All this has fortunately been changed and our schools now, as a rule, furnish satisfactory preparation and degrees of value. But the system of medical education is still defective and does not tend either to economy of administration or to the production of the best and most scientific work. There are one hundred and thirty medical schools in this country which are recognized as worthy to issue accepted diplomas. There are ten in the city of Philadelphia; there are ten in the city of New York; there are eight in Baltimore, and four in Boston. There are sixteen in Chicago whose diplomas are recognized and seven unrecognized. There are nine in St. Louis. In comparison with this it will be found that in the German Empire twenty universities are sufficient; in Austria and Hungary, including Croatia, there are eleven. Manifestly many of the American medical schools could be spared. This is so evident that the medical schools in St. Louis are attempting to combine their energies and to form a consolidated institution. If there is an economy of administration in the union of small railroad corporations, the same principle can be applied to professorships of physiology, anatomy and pathology.

As there is practically no check on the number of medical schools, beyond the good nature of the

* President Eliot as an Educational Reformer: President W. D. Hyde, Atlantic, March 1899, p. 531.

community, the title of professor is not uncommon and its prevalence weakens its authority. The decision of a judge has weight, but the opinion of a professor may not excite even curiosity. Nor can it be said that the country lacks doctors. There are in England 28,000 medical men, in France 17,738 practising medicine. In the United States there are said to be 116,000, or about one to every 600 inhabitants.

This congestion naturally develops competition, which in an intelligent community trains excellent practitioners. England develops great lawyers in spite of a system of education in law so defective as to admit of no defence. It can certainly be said with truth that the medical practitioners in this country will stand comparison with those of any nation. The profession in America is a highly developed one in all that relates to the practical application of medical science. Our hospitals are, perhaps, the best appointed and administered in the world. And it is not only in the larger centres that well-equipped hospitals are to be found. In Massachusetts almost every city or town of enterprise has a hospital furnished and supported in a way to meet all requirements. It is doubtful if the medical needs of a population in so large an area of the world's surface are anywhere so carefully attended to as within the confines of our Republic. *

American contributions to practical medicine

* Hospitals are to be found in thirty-four cities and towns of this Commonwealth, besides the abundant hospital accommodation in Boston.

are well known. Anesthesia, the introduction of abdominal surgery, the surgery of the appendix, reduction of dislocation of the hip, litholapaxy, paracentesis, the development of gynecology, orthopedic surgery, dentistry, the study of fevers and enteric disorders, and the classified studies of functional and nervous diseases hardly need comment.

The American medical profession is certainly characterized by a great readiness to learn and an eagerness for practical knowledge. If the French poet is correct in claiming that the great evil of our time is due to inertia and the great virtue is enthusiasm,* certainly much is to be expected of the American profession, for its enthusiasm is without limit.

The Autocrat has said: "Nothing sheds such light on the superstitions of an age as the prevailing interpretation and treatment of disease, and it may be added that nothing is a better measure of the worth of a profession than its literature. American medical literature was thoroughly provincial for a long period; eagernesss for the teaching of others, however, and a knowledge of personal defects are commendable traits and furnish a good foundation for wisdom. American medical text-books, until recently, were chiefly translations of American issues of foreign books, sometimes with commentaries by the American editor.

* La Princess Lointaine: Rostand. "La grande vice, c' est l' inertie, et la vertue, c' est l' enthusiasme.

An examination of the catalogue of the Harvard Medical School for the year 1875-1876 shows in a list of books recommended to students for text-books and for collateral reading:

| | Foreign. | American. |
|---|---|---|
| Anatomy and histology | 9 | 1* |
| Physiology | 8 | 2 |
| Medical chemistry | 7 | 0 |
| Materia medica | 0 | 3† |
| Pathological anatomy | 4 | 1‡ |
| Therapeutics | 2 | 2 |
| Obstetrics | 3 | 0 |
| Theory and practice of medicine | 2 | 2 |
| Surgery | 7 | 0 |
| Total | 42 | 11 |

In the year 1898-1899 it will be found that there are eighty-five American text-books and seventy-one foreign.

| | Foreign. | American. |
|---|---|---|
| Anatomy | 9 | 8 |
| Histology | 4 | 3 |
| Physiology | 9 | 2 |
| Chemistry | 6 | 5 |
| Hygiene | 1 | 2 |
| Bacteriology | 3 | 3 |
| Therapeutics | 1 | 1 |
| Pathology | 4 | 2 |
| Obstetrics | 4 | 2 |
| Theory and practice | 3 | 6 |
| Clinical medicine | 5 | 8 |
| Surgery | 2 | 9 |
| Gynecology | 3 | 5 |
| Pediatrics | 0 | 5 |
| Ophthalmology | 4 | 4 |
| Otology | 2 | 2 |
| Neurology | 1 | 5 |
| Psychiatry | 4 | 6 |

* A guide for dissection.
† One, United States Dispensary.
‡ A guide for autopsies.

| | | |
|---|---|---|
| Legal medicine | 1 | 1 |
| Clinical microscopy | 0 | 1 |
| Dermatology | 5 | 5 |
| Total | 71 | 85 |

The growth has been greater than would appear from these lists.

At the beginning of the Revolutionary War there were only one book, three reprints and twenty pamphlets by American authors. To-day there are 7,000 books and reprints and a large number of pamphlets. There are 300 books and pamphlets written by American authors annually, and 5,000 journal articles. In 1879-1880, 10,334 books and journal articles were written in the United States in comparison with 7,476 written in England at the same time.* The Americans are probably the greatest consumers of medical literature, and in medical libraries are foremost. The development of medical journals has been remarkable.

Since the latter part of the last century the medical profession has organized medical associations, and there is no healthier sign of the times in American medicine than the growth and excellence of our medical societies. They have raised the standard of education, promoted legislation against quackery; but as the training of the doctor is largely that of a particularist, he is frequently a defective organizer, and our societies often lack in influence which other interests possess. It would be difficult for a State medical society to obtain

* Journal American Medical Association, June 6, 1899, p. 1,214.

from a legislature a grant for the investigation of malaria, but the agriculturists have secured considerable grants for the extermination of the gypsy moth. It is not difficult to raise a large sum for experiments in a new process in the manufacture of steel, on a new application of force, but a doctor would be long a beggar before he could collect funds for a research for the antitoxin of influenza. Our medical societies are too apt to organize more for the relation of personal experience or achievement than for the advancement of an idea. As Dr. Senn has suggested, our societies should become great graduate universities, stimulating and rewarding the highest professional work and influencing the whole body politic.

At the beginning of the century our medical societies were few and small. To day, besides the active special societies and their triennial congress, each State and territory, with but one exception, has its organized society. The national society has probably greater opportunities for influence than any other medical association in the world, and with its membership of ten thousand is perhaps the largest of existing medical associations, and The Massachusetts Medical Society, with its large and increasing membership, has always been a power in the State.

The most distinguished accomplishment of the medical profession in America is the development of the army medical department, which may be regarded not only as a product of our national

existence but of the profession from whose ranks the army medical department is recruited.

When the embattled farmers were gathered at Cambridge in an army which ushered in the Revolution, the second Provincial Congress of Massachusetts Bay, in preparation for the better care of the sick and wounded, in an enactment provided for the thorough examination of all persons asking appointments as surgeons. Thacher, in his military journal during the Revolutionary War from 1775 to 1783, states that the examination which he himself passed occupied about four hours. The subjects were anatomy, physiology, surgery and medicine. A number of the applicants were rejected, and the examination, he says, "was in a considerable degree close and severe, which occasioned not a little agitation in our ranks." The standard which was established was, however, much weakened by the vicious privilege which persists to the present day in our militia, allowing soldiers to choose their own officers and giving the officers in turn the nomination of the surgeon. In the stress of the times the evils of this could not be obviated by any attempts at thorough examinations. The Continental Congress at this time was a body without experience in general legislation, with a fear of centralized authority, and the result was that the Continental Army was absolutely without organization, and suffered from a lack of proper support from the commissary and quartermaster's departments. This chaos was worse confounded by a contest between the regi-

mental and general hospitals, which became so bitter as to prevent any proper administration.

The difficulties were well described in a letter of General Washington to John Hancock, speaking of the improvement required for the hospital department: "No less attention should be paid to the choice of surgeons than of other officers of the army. They should undergo a regular examination, and if not appointed by the director-general they should be subordinate to, and governed by, his directions. The regimental surgeons I am speaking of, many of whom are very great rascals, countenance the men in sham complaints to exempt them from duty, and even receiving bribes to certify indispositions, with a view to secure discharges or furloughs." "We had better trust to the forces of nature and our constitutions than to suffer persons entirely ignorant of medicine to destroy us by ill-directed applications."

The infant nation was, as has been said, "nursed by great men with empires in their brains," and soon the medical department received the careful attention of Washington and eventually seems to have been brought into as an efficient condition as was possible. The exigencies of the service bred a group of unusual medical men whose histories we should be more familiar with. The first in prominence is John Morgan, the first director-general; of him Benj. Rush writes, "As a student he was laborious and painstaking; as a physician learned far beyond most of his contemporaries. As a young surgeon in the British Army he ac-

quired both knowledge and reputation. He may be said to have been the father of medical education in America, for while abroad he elaborated a plan for the institution of medical colleges in the colonies, and he sustained his views in an elegant and scholarly discourse on the subject at the Commencement of the College of Philadelphia on the 31st of May, 1765, and it was by his efforts, aided by Shippen and others, that the college was induced to establish a medical department. The historian who shall hereafter relate the progress of medical science in America will be deficient in candor and justice if he does not connect the name of Dr. Morgan with that auspicious era in which medicine got studied as a science in this country."

Succeeding Morgan as director-general was Dr. William Shippen, Jr., a graduate of Princeton, a pupil of John and William Hunter; he was said by a contemporary to have been "a graceful person of polished manners, and with the tones of his voice singularly sweet and conciliatory. In his intercourse with society he was gay without levity and dignified without harshness and austerity." Under the direction of these men was developed an organization which is noteworthy. The first attempt to check disease in military service by proper attention and ventilation appears to have been devised by Dr. James Tilton, of Delaware, who was in charge of the General Hospital at Trenton, N. J., and who succeeded by a new system of hospital construction in diminishing the sickness resulting from the poisoning of the pa-

tients by the crowding of hospitals. He did away with the hospital tents and private houses then in use, and caused the construction of a large number of log huts, built roughly, so that the air could freely penetrate the crevices. There were no floors, the ground was hardened or baked by heat, and each hut accommodated from five to six men. The fireplace was in the centre and a hole was left at the ridge, permitting the exit of the smoke. The mortality of typhus diminished decidedly in cases treated in this manner. It is also interesting to note that Shippen and Cochran, endorsed by Washington, recommended to Congress a plan of hospital organization, among the provisions of which was the establishment of female nurses in the military hospitals.

Trained in the arduous service of these memorable years were a number of men whose names should not be forgotten: Cochran, Philip Turner, said by Shippen to have been the equal of any operating surgeon in Europe and America; John Jones, afterwards professor of surgery of King's College, N. Y.; James Craik, surgeon in the campaign under Braddock and afterwards the physician to Washington in his last illness; Eustis, later Governor of his State, Secretary of War and Minister to Holland; Brooks, Warren—afterwards professor of anatomy and surgery; Townsend, Hayward, Adams, Homans and many others who left to their descendants a title to hereditary distinction in a plain, printed commission as surgeon in the army, countersigned in the clear, strong signature of George Washington.

The twilight of these surgical demi-gods was devoted after their broadened experience to practice and to the establishment, through teaching, of an American medical profession on the ruins of the destroyed provincial one. They helped to found medical schools and left a tradition of devoted service which has not been lost.

After the establishment of peace, the army medical service was disbanded and as a result of the lack of preparation in military medicine there was great suffering in the War of 1812, that afterclap of the storm of the Revolutionary War. From the confusion which followed there was developed for the first time an organized medical department under the direction of one who should not remain without honor in his own country. Dr. Joseph Lovell, a native of Charlestown, Mass., a graduate of Harvard, entered the army as an assistant surgeon in 1812. Through his abilities he arose to the direction of the army medical department, being the first surgeon-general, as the office was created with his appointment. He served as surgeon-general for thirty years and died in the service, with the respect and love of the whole command. He impressed upon the army medical department, scholarship, thoroughness and efficiency. His report on the diseases incident to camp and campaigning bears the hand-mark of a great medical writer, and was the forerunner of the classical reports issued later from the Surgeon-General's Office.

General Lawson, the successor of General

Lovell, was trained in the Mexican War, and was a man of different mould. He added to the organization great energy and a sense of official dignity which brought the army medical service into the prominence it deserved. It may be remembered that even in the early days, scientific work was carried on by the surgeons of the army. Beaumont's contribution to the study of digestion is too familiar to need quotation. The meteorological reports conducted under General Lawson are perhaps to be regarded as the foundation of the modern science of meteorology. The first observations on the employment of quinine in large doses in malaria also came from the army medical department. The Seminole War, The Black Hawk War, with its cholera campaign, the Mexican War, with its confusion, and the trying frontier experiences, all furnished an excellent school for military surgery. But the whole record of the army medical department from its beginning may be regarded as a preparation for the enormous achievement in the Civil War, that political Krakatoa which shook the continent and sent confusion around the world.

The expansion in two years of a service from the work for a little body of 15,000 men to the greatest medical organization in history for an army of 2,000,000, in a campaign embracing a continent, needed the efforts of so many that it is difficult to select the names of the most important. One of the most forceful influences in the perfection of the service was, in all probability, that

greatest of organizers, Stanton. It is difficult to realize the enormous extent of the medical work of that time. But the medical historian will be attracted by two features: (1) The utilization of the enormous experience for the benefit of scientific knowledge; (2) the establishment of trained services and organizations of relief for wounded and sick.

Woodward's investigations upon the intestinal disorders of the war is one of the great contributions in clinical medicine. The tabulation of the examination of a million recruits is one of the most important contibutions in anthropology ever made, and the "Medical and Surgical History of the War," with its records of cases by the thousands, may be regarded, with the army museum and the medical library, as national monuments testifying for all time to the devotion and thoroughness and high quality of the army medical service in those terrible years.

The medical department was called upon to expend in the four years of war over $45,000,000, exclusive of salaries, for the benefit of the sick and wounded soldiers. Two hundred and one general hospitals were in operation, and a hospital transport service of four sea-going steamers and supplies for 5,000 beds, besides a large number of hospital river boats, hospital railway trains, etc., over 2,000 regimental surgeons, and 85 contract surgeons and 5,500 assistant surgeons. Of these, 32 were killed in battle, 83 died of wounds, four died in prison, seven of yellow fever, three of

cholera, and 271 of other diseases, making a death-roll of 400 names as the profession's life offering in the Civil War.

The enormous number of wounded following the most determined battle of history paralyzed all service, and indescribable suffering followed, but from the chaos, as in the Indian myth of the water-lily rising in the circles of troubled waters from above the pool of decay, there was perfected the greatest organization for the relief of human suffering the world has seen. The establishment of a trained ambulance corps under a system which has served as a model copied by other nations is an achievement sufficient to place Major Letterman on the list of the great benefactors to the race. The Sanitary Commission was the great forerunner of the Red Cross Association, now recognized universally as one of the beneficent associations which relieves war from barbarism.

When the war against Spain came upon the country suddenly it was generally believed by a community ignorant of war that the great efficiency of the medical department in the Civil War could be immediately continued after a lapse of thirty years. It was after the list of sick in the summer camps, and the terrible condition of the Fifth Army Corps at Santiago, and the confusion of the first week at Montauk, that an excited people, inexperienced in the actualities of campaigning, hastily condemned the medical department. Not till the campaigns of this much-photographed and much-written war have been placed

fully in the possession of the community, and the mistakes fully shown, can a critical analysis be made. This will be found to show that the work of the army medical department was not only not culpable, but, considering the difficulties under which it labored, of almost incomparable excellence. It will be found that previous experiences in war were lost upon legislators, and that the army medical department was cut down to the smallest possible point for our small army. It is beyond the possibility of any system to suddenly develop trained and experienced medical officers, and the proper selection of surgeons for an army of two hundred thousand men was impossible. It may be remembered that Surgeon-General Lovell, in 1817, wrote as follows: "An officer of the line may soon learn the duties of the field, and a surgeon be amply qualified for his profession, and both of them be worse than useless in the army. It is from a knowledge of minutiæ, which depend neither upon general regulations nor specific orders, that the experienced officer and surgeon becomes so much superior to the undisciplined recruit. It is almost entirely in order to acquire this kind of knowledge that a military establishment is kept up in time of peace, and it is an undoubted fact that in no department of the army is it so slowly acquired and, therefore, so deficient as in the medical. How severely was this felt during the greater part of the last war (1812) is too well and too publicly known to need comment."

It is in all probability largely due to the neglect

of this common-sense principle that the deaths and the sickness in the summer camps were so great. For the evidence is clear that the sanitation and policing were neglected in the volunteer camp, both by the officers in charge and by the regimental surgeons, those regiments with efficient officers and discipline necessarily suffering from contiguity with ill-disciplined troops. When it is remembered that the marines at Guantanamo suffered no sickness, and that ten thousand troops were encamped at South Framingham without sickness at a time when typhoid and camp disorders were prevalent at Camp Alger, Camp Mead and Camp Thomas, it is clear that an unnecessary amount of sickness and death is to be laid at the score of some one. It would appear to be clear that it is not the army medical department which is to blame.

The campaign at Santiago will probably be recorded by the historian as a modern counterpart of the capture of Louisburg. There was the same gallantry on the part of the men; there was the same successful achievement of the impossible; there was comparatively, judging from the difference in the advance of the science of war, the same confusion. As we now read Parkman's pages, we condemn the mistakes and are proud of the gallantry of the troops, and it is probable that in a century from now, when war has become an exact science, and the art of keeping men efficient, the chief factor in war, has been perfected, the historian will be surprised at the blunders and

incompetency shown by a War Department inexperienced in the perfected science of war.

"Inter arma silent leges" and this is certainly true of the sanitary laws in the hurriedly-prepared campaign of Santiago. We should be led to believe then the American Republic of to-day was incompetent to wage efficient war; but an assertion like this is met by a record strange even in this land of contrasts, namely, the remarkable efficiency of the Philippine expedition, conducted under the same War Department and with volunteer troops. It may be said that after the second week in September order was brought about, well-organized hospitals were established, and medical service brought to a high state of efficiency, of which the profession can well be proud. It is to be remembered, to the credit of the medical department, that if the army was supplied with out-of-date weapons, first-aid packages were furnished to the troops; they were drilled in their application and their beneficient efficiency thoroughly demonstrated.

The country will pardon the sufferings at Santiago as, perhaps, a military necessity, and even condone the bad equipment of the transports and the week of confusion at Montauk. The confusion may be regarded as the result of haste and changed plans, difficult to avoid in war. The mortality statistics show, however, an undue mortality in the training camps. The highest was in August and September, after practical cessation of campaigning, and occurred chiefly in the camps of troops never sent to the front.

COMPARISON OF MONTHLY DEATH-RATES (PER 1,000) FROM DISEASE.

| Months. | 1861-1862. Mean Strength. | Number of Deaths. | Ratio per 1,000 of M. S. | 1898-1899. Ratio per 1,000 of M. S. | Number of Deaths. | Mean Strength. |
|---|---|---|---|---|---|---|
| May . . . | 16,161 | 18 | 1.11 | .26 | 42 | 163,726 |
| June . . . | 66,950 | 55 | .82 | .44 | 90 | 202,526 |
| July . . . | 71,125 | 106 | 1.49 | 1.72 | 451 | 362,613 |
| August . . | 112,359 | 242 | 2.15 | 5.21 | 1,400 | 268,507 |
| September . | 165,126 | 365 | 2.21 | 5.89 | 1,541 | 261,824 |
| October . . | 256,884 | 725 | 2.82 | 3.17 | 809 | 255,000 |
| November . | 301,848 | 1,145 | 3.79 | 1.51 | 365 | 242,000 |
| December . | 343,184 | 1,471 | 4.29 | .84 | 201 | 240,000 |
| January . . | 352,760 | 1,593 | 4.52 | .85 | 180 | 211,000 |
| February . | 327,734 | 1,346 | 4.11 | .87 | 156 | 180,000 |
| March . . | 328,878 | 1,575 | 4.79 | .90 | 123 | 136,000 |
| April . . . | 410,416 | 1,181 | 4.58 | .71 | 80 | 113,000 |
| Annual . . | 229,452 | 10,522 | 45.86 | 25.73 | 5,438 | 211,350 |

But the two thousand deaths in the summer camps will remain as a blot in American history. In explanation many excuses are offered. Training camps badly placed; general officers ignorant or negligent of sanitary laws; appointment of medical officers not sufficiently experienced for their undertaking to important positions; incompetent regimental officers and surgeons appointed by State governors, regiments sent to the camp equipped for war only in their brass bands, ill-disciplined soldiers unacquainted with the elements of sanitary decency, are all mentioned and perhaps all are true. The American people have learned another lesson of the need of thorough preparation, which it is to be hoped will not be forgotten.

The experience of the war has shown the great general ignorance of the elements of sanitation. The medical profession has certainly a large field of endeavor in preaching what has been called the "Evangile of hygienic righteousness."

It is somewhat singular that a nation whose energies have been enlisted chiefly in the pursuits of peace should have contributed so much to the science of war. The rifle was first used by American soldiers at the battle of New Orleans. The open order of attack, hasty intrenchment by the advancing attacking column were productions of American frontier life and are developments of American arms, making war less destructive to human life. If war is, as Shakespeare says, "God's besom" to sweep away the refuse of the world, it will always be needed, and it will be the part of civilization to develop and humanize it. War was originally brute force. Napoleon made it the science of positions, and it should be the duty of our nation, which has done so much to humanize war, to make of it a perfected science of sanitation—the art of keeping men efficient—and the preparation for it a long study of the laws of health.

If the work in the army medical department is more than commendable, that of the navy is superlatively good. In the navy in the last war perfection seems to have been reached; the men not only were as healthy in war as in peace, but in all probability healthier than in the general run of city life. The marines at Guantanamo were

kept in perfect discipline, the springs were guarded and no water was drunk except when sterilized, and although they were exposed to the same influences which overcame the soldiers, there was no sickness. The report of the Surgeon-General of the Navy for the year of the Spanish War shows even better health than in time of peace.

To show what an advance this means, it is only necessary to compare the account of the sickness of the British fleet in the West Indies in 1726: out of a force which never exceeded 4,750 persons, two flag officers, seven captains, 50 lieutenants, 4,000 men died in one year of various forms of sickness.

A statistical comparison of the Spanish War with other wars would be of interest.

The accompanying table prepared by Dr. Mosher, of Boston, of the principal wars of the century shows the relative position of the war in the mortality from disease in the list of other wars. The table shows the percentage of mortality in campaigns by the French and English and Americans in tropical countries.

DEATHS FROM DISEASE.

| | |
|---|---|
| Walcherian (E.), 1809 | 23.33 |
| West Coast Africa (E.), 1824 | 69.00 |
| Mexican (U. S.). 1846 | 10.00 |
| Mexican (U. S.). 1848 | 10.00 |
| Crimea (E.), 1854 | 23.00 |
| China (F.), 1862 | 11.80 |
| Civil War (U. S.), 1862 | 4.00 |
| Civil War (U. S.), 1863 | 6.00 |
| Franco-Prussian (G.), 1870–71 | 1.43 |
| Cape Coast (E.), 1873 | 17.30 |
| Afghanistan (E.), 1878–80 | 9.37 |

| | |
|---|---|
| Egypt (E.), 1882 | 7.21 |
| Soudan (F.), 1883–86 | 28.00 |
| Madagascar (F.), 1895 | 30.00 |
| China-Japanese (J.), 1895 | 1.48 |
| Spanish-American (U. S.), 1898–99 | 2.50 |
| Manila Expedition (U. S.), 1899 | .80 |

TABLE OF MORTALITY FROM DISEASE IN CAMPAIGNS IN TROPICAL COUNTRIES.

| British Army. Name and Date of Campaign. | Died of Disease. Percentage of Strength. | French Army. Name and Date of Campaign. | Died of Disease. Percentage of Strength. |
|---|---|---|---|
| Soudan, 1889 | 0.06 | Tonkin, 1884 | 6.0 |
| Mashonaland, 1876–97 | 0.20 | Tunis, 1881 | 6.1 |
| Suakin, 1885 | 0.22 | Mexico, 1862–63 | 7.1 |
| Soudan, 1885–86 | 0.41 | Tonkin, 1885 | 7.5 |
| China (Talienwan), 1860 | 0.54 | Dahomey, 1893 | 8.7 |
| Ashanti, 1895-96 | 0.56 | Tonkin, 1886 | 9.9 |
| Egypt, 1882 | 0.57 | Tonkin, 1887 | 10.6 |
| [Manila Expedition, 1899, U. S. A.] | 0.80 | Cochin China, 1863 | 10.7 |
| Abyssinia, 1867–68 | 1.21 | Soudan, 1887–88 | 11.6 |
| Galeika Gaika, 1877–78 | 1.40 | Cochin China, 1862 | 11.7 |
| China Field Force, 1860 | 1.49 | China, 1862 | 11.8 |
| Matabeleland, 1896 | 1.65 | Tonkin, 1888 | 13.3 |
| Ashanti, 1874 | 1.74 | Cochin China | 14.0 |
| Zululand, 1879 | 2.48 | Soudan, 1885–86 | 20.0 |
| Spanish-American, 1898–99, | 2.50 | " 1886–87 | 22.0 |
| Chitral, 1895 | 2.51 | " 1884–85 | 22.5 |
| Nile, 1884–85 | 3.64 | " 1883–84 | 28.0 |
| Dongola, 1896 | 4.66 | Madagascar, 1895 | 30.2 |
| Afghanistan, 1878–80 | 9.37 | | |

The English campaigns show figures that are better and figures that are worse than those of the Manila campaign. Their mortality in one tropical campaign was down to 0.06 per cent. In contrast to this the English mortality in

the Afghanistan War ran up to 9.37 per cent. and to-day averages in India 4 to 6 per cent. at a time of peace. Kitchener's Soudan campaign would seem to offer the most recent chance for comparison. When, however, the conditions of the two, the Soudan campaign of Kitchener and our Santiago campaign, are examined, they are found not to be comparable. For instance, the English troops were mostly seasoned natives, plus a small acclimated European contingent. Our men were green troops, not veterans, and anything but acclimated. The English had the same heat to contend with that our troops had, but they did not have the tropical rains — nor did they fight in a yellow-fever country. Notwithstanding all the advantages on the side of the English, typhoid broke out among them and followed them, and "the mortality during the campaign from enteric fever . . . will probably be found, when full reports are in, to be excessive." * One battalion lost 16 per cent., "a formidable percentage, fully equalling that of some of our green volunteer regiments on their return from Cuba, where they had the special disorders of malaria and yellow fever also to combat."

So far in the Manila expedition the losses from disease are 0.8 per cent. In 1824 the English lost from disease on the West Coast of Africa 69 per cent. of their men, "so that it became necessary to enumerate the strength each quarter, as the troops were cut off with such rapidity that

* Journal of American Medical Association, December 10, 1898, p. 1,426.

few lived to complete one year in the command." At most one per cent. of our men died of disease in 1898, while during the years 1862–1865 4 to 6 per cent. died. In other words, to quote from Department Surgeon-General Smart: "Thirty-seven years ago the flower of the manhood of this country, after months of deadly losses, suffered in April, 1862, more than twice the loss incurred in August last (our worst month), and, instead of going home on sick furlough, girded up their loins for the attack on Richmond by way of the Peninsula."

The other noteworthy figures of the tables are the figures of the American wars by themselves. There is a steady decline in the loss of death by disease. Ten per cent. died of disease in the Mexican War; 5 and 6 per cent. in the Civil War, and 2.5 per. cent. in the Spanish-American War, and 0.8 per cent. in the Manila expedition. The mortality and sickness in the Austro-Prussian War was high, but the small loss in the Franco-Prussian War, 1.42 per cent., is noteworthy, though it should be remembered that the campaign was not one of great hardship; but especially striking are the tremendous losses of the French in the Soudan campaign, 28 per cent., and in the Madagascar campaign, 30 per cent. These figures of the French losses were taken from a table comparing the losses of the French and English troops in recent campaigns in tropical countries. The table showed that with the exception of the Afghanistan campaign of the English, which had a mortal-

ity of 9 per cent., due to an outbreak of cholera, the most unhealthy of eighteen English campaigns was healthier than the healthiest of eighteen French campaigns.

The most unhealthy English was Dongola, 4.66 per cent., and the healthiest French campaign was the Tonkin campaign, with a mortality from disease of 6 per cent. This fact and the tremendous mortality of the Madagascar campaign, 30 per cent., led to an investigation.

This table shows, then, that the losses from disease in the Santiago campaign were relatively small, and that the losses of the Manila expedition will compare with the best. Our losses from disease in the field are open to but little criticism. Men, however, died from disease not in the field but in the camps. Is our mortality open to criticism? The commission appointed by Surgeon-General Sternberg to examine this point reports that it is. The words of Dr. Vaughan are, "a mortality of 7 per cent. in camps which should be hygienic is rather high." "This could have been prevented had the Government been more careful in the selection of volunteer surgeons."

Further on in his paper on typhoid in our army camps he says: "I meant that the average volunteer surgeon was better than the average doctor in civil life. The point of the matter is that the medical officer is powerless unless the line officer will uphold him and follow his directions. . . . I think it is a shame and a crime that the line officers

of our regular army have no education as to sanitary knowledge.

"How did the disease (typhoid) spread? There is no evidence that the water supply in the larger camps was infected. . . It may be said truthfully that the spread of this disease was caused by the improper disposal of excreta. The epidemic was not due to sending northern men to a southern climate; it was not due to the locality or to the massing of men in one place, but it was brought about by camp pollution. The men's feet carried the excreta, flies carried it to food, and thus it was spread."*

The high mortality of our summer camps was due to poor policing and poor hygiene; one the fault of the army, the other the fault of defective sanitary education. The mortality of war does not depend upon missiles so much as upon neglect in sanitary care. The rifle of the enemy is not as deadly as neglected sinks and foul hospital bedpans.

In comparison with this the mortality of the Spanish Army in Cuba, 1897, is important.† The following figures are approximately correct for this year, 1897.

| | |
|---|---|
| Deaths from yellow fever | 6,034 |
| Deaths from enteric fever | 2,500 |
| Enteritis and dysentery | 12,000 |
| Malarial fevers | 7,000 |
| All other diseases | 5,000 |
| | 32,534 |

* Medical Record, New York, May 13, p. 683.

† Medical News, New York, lxxii, 592; W. F. Brunner, M.D. Sanitary Inspector, report to Surgeon-General.

"I can safely say that 10 per cent. of the 30,000 invalided home were destined to certain and early death. These enormous death-rates, it must be remembered, occurred in an army which at no time was properly cared for, and was always badly clothed and badly fed; exposed to all the dangers of a tropical climate, they succumbed easily."

Sick percentages of the United States Army from 1840 to 1899 show marked improvement in the development of sanitary science.*

| | | |
|---|---|---|
| 1840–1854 | 8.9 | per cent. |
| 1862–1866 | 20.00 | " |
| 1867–1897 | 4.5 | " |
| 1898 (entire army) | 3.5 | " |
| 1899 (Manila) | 8.9 | " |

In regard to the health of the troops in Manila, General Otis writes in April:†

"I think that the health of the command will compare favorably with the British troops in India, there being only about 8 or 9 per cent. on *sick report* from all causes. The *mortality* of the British Army in India averages from 4 to 6 per cent., so that the percentage of sickness must equal, if not exceed, the percentage of sickness of our troops in Manila."

The philosopher of Chicago, viewing this country from behind his bar, shows contentment when he pronounces that "This is a great country, and the best of it is that we know it," but that the medical profession may not fall into

* Journal American Medical Association, April 22, 1899, p. 873.

† Journal American Medical Association, April 22, 1899, p. 873; Forum, May, 1899, p. 345.

that condition of self-congratulatory complacency which is often the initial stage of degeneration, our sanitary statistics may serve as a corrective.

In 1896, 75,000 deaths occurred in this country from typhoid. In 1898, 1,713 deaths from typhoid occurred in three months in Philadelphia.

Typhoid fever is, as is well known, not only a preventable disease, but it is prevented as shown by the statistics of Munich and Hamburg; and Berlin, formerly the abode of filth, now the cleanest city in the world, is now almost free from typhoid. A few statistics will make the facts of our sanitary deficiencies more clear. (See page 34.)

The accompanying table showing the death-rate from typhoid fever in Massachusetts indicates how impure water supply is related to the disease:

SUMMARY BY DECADES, 1856–95.

| Period. | Death rate from typhoid fever per 100,000. | Percentage of population not supplied with public water. | Period. | Death-rate from typhoid fever per 100,000. | Percentage of population not supplied with public water. |
|---|---|---|---|---|---|
| 1856–65 | 92.9 | 75.44 | 1876–85 | 47.4 | 31.75 |
| 1866–75 | 80.8 | 58.94 | 1886–95 | 36.4 | 13.93 |

It will be seen that Boston is higher in typhoid death-rate than Amsterdam, Berlin, Brussels, Edinburgh, Glasgow, Hamburg, Liverpool, London Munich, Paris, Vienna, and if this is true of Boston, what is to be said of Philadelphia?

DEATH-RATE FROM TYPHOID FEVER PER 100,000 POPULATION PER ANNUM, 1890–1895.*

| | 1890 | 1891 | 1892 | 1893 | 1894 | 1895 | 1896 | 1897 |
|---|---|---|---|---|---|---|---|---|
| Albany | 60 | 108 | 50 | 59 | 52 | 165 | | |
| Amsterdam | 19 | 11 | 15 | 16 | 8 | | | |
| Atlanta | 149 | 119 | 87 | 66 | 43 | | | |
| Baltimore | 57 | 34 | 42 | 47 | 49 | 28 | | |
| Berlin | 9 | 10 | 8 | 9 | 4 | | | |
| Boston | 32 | 33 | 25 | 26 | 23 | 32 | 33 | 32 |
| Brooklyn | 21 | 21 | 18 | 18 | 16 | 16 | | |
| Brussels | 26 | 41 | 23 | 27 | 14 | | | |
| Buffalo | 44 | 56 | 38 | 37 | 62 | 28 | | |
| Cambridge | 16 | 21 | 20 | 21 | 30 | 15 | 36 | 15 |
| Chicago | 83 | 160 | 103 | 42 | 31 | 32 | | |
| Cincinnati | 67 | 62 | 40 | 43 | 50 | 36 | | |
| Cleveland | 66 | 52 | 54 | 47 | 27 | 36 | | |
| Copenhagen | 9 | 8 | 7 | 9 | 7 | | | |
| Detroit | 18 | 13 | 51 | 61 | 26 | 24 | | |
| Edinburgh | 19 | 18 | 13 | 14 | 15 | | | |
| Fall River | 53 | 66 | 38 | 20 | 33 | 33 | 30 | 45 |
| Glasgow | 26 | 31 | 18 | 20 | 24 | | | |
| Hamburg | 28 | 23 | 34 | 18 | 6 | | | |
| Hartford | 60 | 73 | 82 | 50 | 51 | 58 | | |
| Lawrence | 134 | 119 | 105 | 80 | 47 | 31 | 19 | 16 |
| Liverpool | 24 | 25 | 25 | 53 | 58 | | | |
| London | 16 | 15 | 11 | 16 | 15 | | | |
| Lowell | 160 | 96 | 90 | 62 | 60 | 39 | 40 | 19 |
| Montreal | | | | 20 | 20 | 23 | | |
| Munich | 8 | 7 | 3 | 15 | 2 | | | |
| New Haven | 28 | 20 | 29 | 31 | 26 | 34 | | |
| New Orleans | 20 | 23 | 21 | 15 | 28 | 41 | | |
| New York | 21 | 22 | 14 | 20 | 17 | 17 | | |
| Paris | 30 | 20 | 28 | 25 | 29 | | | |
| Philadelphia | 64 | 64 | 34 | 41 | 32 | | | |
| Pittsburg | 131 | 100 | 100 | 111 | 56 | 77 | | |
| Richmond | 88 | 60 | 68 | 53 | 31 | 27 | | |
| Rome | 35 | 36 | 26 | 34 | 30 | | | |
| San Francisco | 45 | 34 | 32 | 34 | 37 | 32 | | |
| St. Louis | 34 | 30 | 37 | 103 | 31 | 19 | | |
| Toronto | 80 | 90 | 40 | 40 | 20 | | | |
| Vienna | 9 | 6 | 8 | 7 | 5 | | | |
| Venice | 44 | 33 | 30 | 26 | 18 | | | |
| Washington | 89 | 86 | 72 | 72 | 72 | 69 | | |
| Worcester | 17 | 21 | 18 | 33 | 32 | 15 | 26 | 15 |

* Prepared by the kindness of the Massachusetts State Board of Health.

Another means of indicating the medical intelligence of the community is found in the figures of infant mortality.*

INFANT MORTALITY IN DIFFERENT COUNTRIES.
*Deaths under One per One Thousand Births.*

| Countries. | Years. | Infant mortality per 1,000 births. | Countries. | Years. | Infant mortality per 1,000 births. |
|---|---|---|---|---|---|
| Ireland . . | 1884–88 | 94 | Italy . . . . | 1884–91 | 192 |
| Sweden . . | 1881–90 | 97 | Prussia . . . | 1886–92 | 207 |
| Scotland . . | 1885–90 | 120 | Hungary . . | 1884–97 | 212 |
| England . . | 1885–91 | 144 | Austria . . . | 1886–87 | 246 |
| Belgium . . | 1881–91 | 159 | Saxony . . . | 1386–92 | 281 |
| France . . | 1885–90 | 165 | Bavaria . . . | 1879–88 | 287 |
| Holland . | 1885–90 | 179 | Massachusetts . | 1876–95 | 161 |

As Dr. Abbott says, "From this table it appears that the infant mortality of Massachusetts is greater than that of Sweden, Ireland, Scotland, England and Belgium, but less than that of the other countries named in the table, that of Bavaria being greater than our own by 78 per cent."

State sanitation has been brought in Massachusetts to a high state of excellence through the efforts of Drs. Bowditch, Derby, Folsom, S. W. Abbott and our own former president, Walcott. The work of the Massachusetts State Board of Health is to be compared with the best existing. This is also true of many of the boards of health of our cities, especially that of Boston under Dr. Durgin, but unfortunately only to a limited degree

* Infant Mortality in Massachusetts, S. W. Abbott, M.D., Journal of the Massachusetts Association of Boards of Health, vol. viii, No. 4, 1898.

of the State boards, and largely owing to the lack of proper legislative support. In the great State of Pennsylvania, the last grant for the Board of Health was only $8,000, exclusive of salaries of officials, for the coming two years' work. In New York State, with its enormous wealth, the last annual appropriation was nearly one-half less than that of Massachusetts, only $30,000, and in most of the States the regular appropriation granted is barely sufficient to continue the work of the regulation of the practice of medicine necessarily assumed by the boards of health.

The importance of sanitary work can not be over-stated, and as it is clearly owing to a lack of public knowledge of the facts that it is defective in America, it is the duty of the medical profession to call attention to the matter. Obstacles may be imagined in the lack of interest on the part of the spoilsmen politicians — those whom Bismarck classifies as *novarum rerum cupidi* — but only eager for a change for their own advantage. Politicians, however, even at their worst, are obliging tyrants, for they are certain to yield actively to determined public opinion. It is not difficult to awaken popular interest in improvements in laws relative to industrial hygiene, that is, the health of working people, in their various trades, in which we are behind both Germany and England, in the establishment of contagious hospitals in which we are not as well supplied as in England, and in sanitaria for phthisis, in which America is less well advanced than Germany.

The importance of earnest efforts for the checking of tuberculosis is well expressed in this circular distributed by the German Bureau of Health:* "Some estimate may be made of the financial benefit which such public hospitals for consumptives would bring to the people by the following calculations: Assuming that only one-seventh or one-eighth of the 90,800 persons between the ages of fifteen and sixty who die annually in Germany from tuberculosis — or in round numbers, 12,000 — were subjected to treatment, and that of these three-fourths, or 9,000, were restored to health or ability to work, and thus remained alive and with their families for three years longer than would be otherwise possible; and estimating the wages of a man (in Germany) to be 600 marks ($150) a year, or say an average of 500 marks ($125) for each person between the ages of fifteen or sixty, then there would be a total gain of 3 x 600 x 9,000, equals 13,500,000 marks ($3,375,000), for those who were thus benefited. To do this would require an expenditure, say, of 400 marks ($100) for each of the 12,000 persons treated, without deducting interest on plant, or a total cost of about 5,000,000 marks ($1,250,000). If now the interest on the estimated capital necessary to maintain 4,000 beds be added to this, or 1,000,000 marks ($250,000) more, there would remain, after subtracting the total cost of 6,000,000 marks ($1,500,000) for the annual treatment of 12,000 patients, a total yearly gain to the public of 7,500,000 marks

* From Circular from the Kaiserliches Gesundheitsamt.

($1,875,000). To this should be added the profit to the patients themselves in the pleasure of living, and the protection afforded to the children and families of those restored to health, to whom is thus preserved the support of the working father or care of the devoted mother for perhaps many years to come."

Our position in regard to mortality from phthisis is shown in the accompanying table, and also the need of strenuous endeavor to place ourselves in the front rank of public health. It is true that climate is a factor in the matter of tuberculosis, but rational treatment, proper environment, is probably of greater importance, and already sufficient gain has been shown to stimulate efforts which should place us with our wealth in the highest instead of the lowest place.

DEATHS FROM PHTHISIS AND PULMONARY TUBERCULOSIS PER MILLION INHABITANTS.

*Average for Years 1887 to 1893 and 1894.*

| | | |
|---|---|---|
| Prussia | 2,715 | 2,389 |
| Massachusetts | 2,518 | 2,230 |
| German Empire | 2,428 | 2,363 |
| Ireland | 2,124 | 2,092 |
| Scotland | 1,794 | 1,723 |
| England and Wales | 1,568 | 1,385 |
| Italy | 1,340 | 1,303 |

DEATHS FROM TYPHOID FEVER PER MILLION INHABITANTS.

*Average for Years 1887 to 1893 and 1894.*

| | | |
|---|---|---|
| Italy | 688 | 442 |
| Massachusetts | 380 | 310 |
| Ireland | 228 | 205 |
| Prussia | 215 | 151 |
| Scotland | 194 | 176 |
| England and Wales | 191 | 166 |
| German Empire | 165 | 131 |

DEATHS FROM SMALL-POX PER MILLION INHABITANTS.

*Average for Years 1887 to 1893 and 1894.*

| | | |
|---|---|---|
| Italy | 293 | 85 |
| England and Wales | 17 | 27 |
| Prussia | 3 | 2 |
| Ireland | 1 | 16 |
| Massachusetts | 2 | 13 |
| Bavaria | 2 | 0.3 |
| Saxony | 2 | 1 |
| Scotland | 5 | 21 |
| German Empire | 3 | 2 |

DEATHS FROM DIPHTHERIA AND CROUP PER MILLION INHABITANTS.

*Average for Years 1887 to 1893 and 1894.*

| | | |
|---|---|---|
| Prussia | 1,456 | 1,473 |
| German Empire | 1,383 | 1,308 |
| Massachusetts | 721 | 740 |
| Italy | 665 | 506 |
| Scotland | 405 | 443 |
| England and Wales | 307 | 350 |
| Ireland | 228 | 201 |

In regard to one disease the public sanitary sense appears to be excellent, namely, small-pox, and the marked improvement in mortality from diphtheria and croup in this city indicates sanitary and medical efficiency.*

We are told that "righteousness exalteth a nation: but sin is a reproach to any people." Sin can be interpreted as disobedience to the laws governing the universe. Where these laws are known the prevalence of disease becomes both a reproach and a national crime. It is the duty of our profession, of which it may be said, "Altius caderis Dei patefacit arcana," to study and teach

*The mortality in Boston from croup and diphtheria has been brought through the efficient efforts of the Board of Health to as low a rate as that in England.

these laws. Is this done in America as it should be? In Germany there is an Imperial Health Office (*Kaiserliches Gesundheitsamt*). In England the Local Government Board regulates matters of health. In France the Council-General of Health (*Conseil générale de Santé*) is consulted on national health. Even in Italy there is a Hygienic Institute for advice. In America there is no national health commission, board or bureau. At the Congress of Tuberculosis, scientific American medicine was represented by a doctor of the Biochemic Department of the Agricultural Bureau at Washington. Should not our National Medical Association or our State societies, which have accomplished so much in improving the standard of the profession and of medical education, address themselves earnestly to the task of removing from our country the reproach of neglect of the highest functions of government, or from being what Dante terms,

"genti dolorose
"ch' hanno perduto 'l ben dello 'ntelletto." *

"An unfortunate race, who neglect the help of knowledge in sanitation," as it may be paraphrased.

This suggests an inquiry as to the worth of American contributions to the science of medicine as distinguished from the practical application of knowledge previously obtained. What has America contributed besides confirming, improving, adding to what has been discovered or taught by others?

* Inferno III, Canto III.

When the record of this remarkable century which is ending is looked at from a sufficient distance for a proper estimate of values, the acheivement most beneficial to humanity will perhaps seem to be the lifting of the art of medicine from the level of an unsatisfactory branch of metaphysics, vivified by empiricism, to the level of the science with the greatest promise to human welfare. Montaigne and Molière were not much in error in their ridicule of the medicine of their day. The empiricism of Chinese and Thibetan medicine, formulated for centuries in aphorisms, loaded with demonology and spirit worship, are not more tedious than the writings and discussions of two centuries ago, full of discussions as to the humors, the mineral, vegetable and vitalistic theories of disease. The advance from this to the science of disease of to-day is, in the last one hundred years, chiefly the work of France and Germany. The work may be grouped as: Pathological research, by Cruveilhier, Rokitansky, Virchow and his pupils; improved methods of clinical observation and record, introduced by Corvisart, Lænnec, Louis and bis followers; the great triumph of Pasteur in his demonstration of the influence of micro-organisms in the causation of disease; and its corollary the protection and cure by attenuated virus, followed by the discovery of specific germs by Koch, his co-workers and followers; later the study of bacterial poisons and the cure by serumtherapy, developed by Behring, Roux, Ehrlich.

Lister laid humanity under the greatest debt by the application of the germ theory to surgery, and saved to the English-speaking race the credit of a share in the magnificent medical triumph of the century. It is impossible to associate American medicine largely with these achievements. It is difficult to connect an American observer with the discovery and demonstration of the germ of a human disease. In the study of the bacterial poisons, out of eighty-nine observers mentioned in an American text-book on the subject, but six are Americans, Italy contributing twelve and Japan four. Leidy's discovery of the trichinæ in the hog and Jeffries Wyman's confirmation of Pasteur's investigations were of much value, but hardly more than contributory. The discovery of anæsthesia was of inestimable value, but although it is a source of national pride, it can hardly be considered an effort of scientific medicine; it was an achievement of magnificent empiricism.

As if to show no absence of true scientific spirit in this country, the work of Welch, Councilman, Sternberg can be mentioned, and of Vaughan, Novy, Chittenden and Minot, Bowditch, Howell, Porter, but especially the fruitful investigation of Theobald Smith. The work on the Texas cattle-plague was conducted under the direction of the Department of Agriculture, but its value to medicine is not limited to cattle.

The demonstration that certain diseases are conveyed by insects, the discovery of the germ, the investigation of the development in the intes-

tine of the insect-carrier and of the transmission of the disease from the insect to a second animal, are achievements of the highest importance, and have been the forerunners of the important work on malaria in man, by Laveran, Ross and Grassi, a work, however, which is largely to the credit of French, Italian and English investigators.

It has been said so often that allowances should be made for us, a young country, that the truth of this has generally been accepted among us, forgetting the underlying fallacy of the statement. The founders of the country were not savages, but selected men, bringing the traditions of advanced civilization. It was said that "The Lord winnowed three kingdoms to plant New England." The early limitations were those of poverty, not of inclination, and poverty has been overcome among us for nearly a century. Our oldest university is nearly three centuries old. It has also been said that the genius of the American people is practical and not theoretical, and that the gift to humanity to be expected of us is to be in contributions to human physical welfare rather than to thought or research — an opinion more calculated to satisfy our national complacency than to add to our national influence; nor does the opinion stand the test of examination.

The growth in material prosperity of the North American Republic has been so extraordinary that it is not strange that it should overshadow advances in other directions. But if material growth has been our only achievement we must admit that our

civilization is as yet incomplete or a failure, for "That society," says Emerson, "can never prosper, but must always be bankrupt, until every man does that which he was created to do." Is it true, however, that there has not been remarkable advance in scientific pursuits in this country?

In astronomical investigation the work of this country is in the first rank; the science of meteorology may be almost claimed as a national product; as in America it was first placed on a scientific basis, and it is in this country that its application is most general and most appreciated. Paleontology, geology, ethnology, biology and botany have in our American universities representatives of the highest rank. The archeological work of North America has been naturally more directed toward the archeology of this continent, but the recent researches in Chaldea and Babylon, as well as in Greece, have been of such excellence as to be universally recognized.

It can certainly not be claimed that the country is incapable of scientific effort. And if this is granted, and there is no lack of ability in the medical profession, the lack of achievement in this country in research in the medical sciences is evidently a defect. If the bridge over the Nile at Atbara is given to Americans because they are the best bridge-builders in the world, and if more than half the new stars discovered in this decade have been found by American observers, why is it that in the recent scientific investigations which have revolutionized medicine — the discovery of

germs, toxins and antitoxins — America has taken almost no part? It is not that there is lack of energy or of ability in our profession or of wealth in our communities. The difficulty lies simply in the fact that the profession has not yet addressed itself earnestly to the problem of scientific medical research, but has been contented with the training of medical practitioners. So much is this true that the defect has hardly yet been realized, for as common-sense in the practitioner is the ideal striven for by the profession, so scientific research has been left to the efforts of others. Common-sense was of value in the days of theoretical speculation. But of what avail is common-sense as a help to a patient dying of cancer, or tetanus, or hydrophobia? The world to-day is waiting for new research. Is the American profession to remain behind? The remedy for this condition is simple and lies with the profession. The public should be informed of the needs of the time — a public which has never withheld financial support where the want has been shown. Well appointed and well supported laboratories for medical research are needed in every community, at every medical school and every hospital. Our medical schools should be organized for a greater purpose than granting diplomas or licensing young practitioners of medicine.

America is destined to be the great distributor of the world's products — natural and manufactured; it is certain to be blessed with wealth almost beyond limit, but as our great preacher has

said, "God's gifts do not measure our worth but our responsibilities," and it is a serious question how our debt to the world is to be paid.

When the fleet at Cavite fired the volleys heard around the world, unexpected anxieties unnumbered came upon the country, but one great benefit will follow if the nation is brought to a sense of responsibility to the highest standards. We are no longer a young country to be judged apart from the rest of the world — we are to be measured by what is expected of us. This, from a land of our resources, will be the greatest gift to human welfare possible in human effort. Nothing less than this will save us from the stigma of shortcoming. There are difficulties in rising to the level of our opportunities, but

> "Steep and craggy is the path of the gods,"

and this high standard is held up to us in art, in science, as in commerce and in power.

America has been said to breed

> "A race of victors,
> A race ready for conflict, the race of the conquering march."

The conflict of the next century will be against ignorance, sorrow and suffering, and in this the medical profession must be foremost in strenuous endeavor.

ARTICLE II.

THE SHATTUCK LECTURE.

# NOT THE DISEASE ONLY, BUT ALSO THE MAN.

By JAMES J. PUTNAM, M.D.
OF BOSTON.

DELIVERED JUNE 13, 1899.

# NOT THE DISEASE ONLY, BUT ALSO THE MAN.

Mr. President and Fellows
of The Massachusetts Medical Society:

I take as the text for my address the familiar sentiment that often falls so solemnly from the lips of older members of the profession in addresses to students who are about to graduate from the schools of science into the school of life: "Remember, when you go to see your patients, that it is after all the man, not the disease, that you are called upon to treat."

Impressive as this phrase sounds, I have often wondered just what the various speakers had in mind. This patient might be poor in purse, that one predisposed to certain ailments; the social or domestic surroundings of a third might make his case a peculiar one, and so on. But such external circumstances, important as they might be, would not exhaust the significance of the admonition. We cannot really know the man whom we are called upon to treat without going far beyond his outward relations and penetrating in imagination deep into his mental life. "The man" is, above all else, the mind of the man, and not only the mind as an organ of conscious thought but the mind as an organ of bodily nutrition, and the mind as a vast theatre for the interplay of contending forces that do not always recognize the personal consciousness as their ruler. This is the man that the doctor should learn to know

and treat. The need is not met by kindliness and humanity alone; the task is one for science also. Yet medical schools do not prepare their students at all adequately for this duty, or include a knowledge of psychology among their requirements for matriculation. The genius of the truly great physician may bring him to the goal, it is true, without a master's guidance. But if we are not content with the intuitions of the few; if we wish to see principles laid down that shall reflect the highest possibilities of medicine, the training of the doctor must begin during college life. I should rate a thorough preliminary course in psychology and philosophy far above a knowledge of botany or zoology, and as following close on chemistry and physics, as a preparation for the work of a general practitioner.

It is not the least of the benefits that will come to us from the insistance on a college degree as a condition for entrance to the Harvard Medical School, that a better chance will have been afforded for remedying this lack in the students' fitting. We want to see, in the men whom our university sends forth proud of her warrant of tasks faithfully performed, not only sporadic signs of a capacity to recognize and control, each after his own fashion, the gigantic and ever active forces of the mind, but abundant evidence that this problem has been, in all its many aspects, an object of their studious thought. The physicians who have previously been good students of psychology have, in my experience, proved themselves to be men of unusully broad sympathies and high ideals, and keen to see causes of disease to which the pure pathologist is often blind. For what a man sees or fails to see depends, as we all know, on how the vast storehouse* of his mind has previously been stocked. The in-

* I do not think it really accurate to speak of the brain as a "storehouse," as is so often done, though the term is familiar and expressive. In fact, the brain is no more a storehouse of the events that have transpired in and through it than the brass disk is a storehouse on which the sand will shortly arrange itself, at the touch of the bow, in the wonderful Lissajou's lines, or than the sounding board of a piano is a storehouse. When the

habitants of that hidden world, like groups of men in society, welcome the new-comer whose manner and dress are familiar, and let the others pass unnoticed.

No argument is needed to show what transforming power the mind may exert. The energy set free by the magic agencies of hope, courage, desperation, fanaticism, or by the enthusiasm for a great cause, may reveal the possession of a force undreamed of, or so husband the resources of the body as to keep the flame of life burning for a time when the oil seems exhausted. These great influences have been the doctor's best ally since time began; the harp of David singing to Saul is heard again in the music which is used to tie with new bonds to life the inmates of our State hospital for the insane at Danvers. The great leaders of the scientific centuries to which we belong have indeed turned a cold shoulder on the time-honored methods of cure by faith and fear which had worked miracles for ages past. Their work lay in establishing the reign of exact methods of inquiry, and they would tolerate no systems that rested on a less firm basis. But the movement which the labors of these men of science inaugurated is now secure beyond all hazard, and the time has come to recognize that there are others that may justly lay claim to our attention.

brain is inactive (if one could imagine such a state) it is incapable of telling any story as to its history. But each new stimulus revives each old activity, of identical, or similiar, or congruous, or contrasting sort, and so on. The reviving flash of consciousness which occupies each new moment, like the thrill which at the same moment sets the brain into vibration, is a single complex impulse, an instantaneous photograph, and although it takes the form of memory, it belongs in fact wholly to the present.

Neither does it seem to me exact, at least from the point of view of the cerebral physiologist, to speak of "inheriting ancestral experiences." What we inherit is a brain so plastic in certain directions and so little inhibited on those lines that it organizes its experiences of certain sorts with great rapidity.

The power which imposes as "intuition," and which seems endowed with faculties that seem to transcend and antedate experience, may, I think, be, after all, experience—taught, though the teaching is marvellously rapid and effective. Any one of us, if he could have appreciated and appropriated and made his own all the opportunities that have been afforded him for education of the brain in any special line would be a genius in that direction.

We may accept the limitations which the scientific method imposes, even to the point of sacrificing intuition for the sake of a more conscious insight. But we should not admit any arbitrary limitation of the field of inquiry, nor rest content until scientific research has taken all the divinations of instinct into account. The splendid wave of pathological and bacteriological research that has raised the art and science of medicine to its present position of authority has set physicians trying to solve their problems in terms of pathology alone; but it is a matter for congratulation that this wave is being reinforced by another which is sweeping us toward a better knowledge of the secrets of the mental life in health and disease. Perhaps we may even learn thereby to interpret the teaching of pathology itself in a wider sense.

The physician is an educator; the successes of the school-master are his successes; the fact that even after such long years of scrutiny and experience the pedagogue still finds new methods of impressing his pupils, and new principles to guide him, gives warrant for the hope that the same may prove true for the physician, provided only that he brings the same amount of patient labor to the solution of his special problems. I doubt if most general practitioners are aware how far, in fact, this hope is already realized. We all hear of the triumphs of surgery, and every doctor feels a personal pride in spreading the news of them abroad. But there are not so many who know that there are men, as yet few in number but of equal genius, who with equal devotion and study have been searching for means to change, little by little, the trend of the forces that work within the mind, and are thus silently laying the foundations of a new departure in therapeutics. This is education of a high order,*

* I will refer, simply by way of illustration, to the recent searching analysis of actual cases by the now well-known Pierre Janet. [*Névroses et Idées fixes, Paris*, 1898.]

The following paragraph [p. 406], which concludes one of the chapters,

and such studies as these must yield conclusions that the well-trained doctor will find use for in his daily practice.

In the sort of training that is undertaken for the sake of breaking up morbid habits of the nervous system, or of establishing favorable habits under difficult circumstances, the need of inducing reactions that shall have an effective outcome is even more evident than where only the teaching of the school-room is at stake. Definite accomplishment, and motion at any rate, are often the prime conditions of mental health. If water does not flow it stagnates, and the more so in proportion to its lack of purity. To this outcome of effectiveness, the effectiveness which implies continuous growth through utilization of evil and of good, the influence of the doctor, above all other educators, is instinctively pledged. No wonder that his efforts are often crowned by disappointment and failure; these are but the disappointments and failures of the school-room intensified.

Fortunately, no one here is likely to interpret too narrowly the meaning of effectiveness. There is absolutely no criterion of accomplishment except that which should say whether or not a man had done his best. To refrain from acting is often the fullest act. Washington coolly facing his enemy and his own limitations, and waiting for the chances that rarely came, presents no figure of greater energy or heroism than is presented by the resigned, the cheerful, the ever-ready neurasthenic patient who is doing his best. "They also serve who only stand and wait."

The doctor is most obviously an educator in dealing with the training of children, when the task that he sets for

may give some conception of the author's aim and spirit: "Objective psychology [*i.e., by clinical observation*] does not aim at supplanting subjective psychology, which will remain indispensable for the study of the laws of thought. Neither does it aspire to set up any system of metaphysics or religion, or to overthrow any earnest belief. Its task is to gather together the facts which are essential for a real insight into the working of men's minds, and to discover the art of securing the freest play for healthy mental life and the best alleviation for mental disease."

himself is practically identical with that necessarily set for the school-master and the parent. I refer to this class of cases for the sake of calling attention to the remarkable results now attained at the various institutions for defective children in this country and in Europe, among which our Massachusetts School for the Feeble Minded, under the able leadership of its present superintendent, might compete for the highest rank. So fine are the chances thus presented that the definition of feeblemindedness is now being widened to secure the benefits of this training for children not heretofore recognized as defective. Even when regarded simply as schools for the training of teachers and the development of methods of teaching, such institutions as this are bound to hold an exceptional place, because in order that any success at all should be gained, the slightest action and reaction of each pupil's mind have to be recognized, while the educational principles that are followed must be of the best sort, and must be administered with personal devotion and close attention to details.

Even where the child is not strictly defective, but simply a hard case for the parent and the school-master, the methods of influence to which clinical research has given birth are likely to find an important if not a wide field of usefulness. The subject is too large a one to speak of in detail, but there are cases, as the teacher should realize, where the birch and the fool's cap are less useful than the training by the aid of influences such as I shall presently refer to, to be administered under the skilled supervision of the doctor alone. Certain disorders of the physiological reflexes, such as enuresis, are eminent examples of an analogous though a more obviously medical sort. There are cases of this kind where even hypnotic suggestion, of a mild degree and such as any physician can soon learn to use with safety and sufficient skill, is the best cure. In fact, the only rational treatment of enuresis and constipation consists in a re-training of the

physiological reflex, and the re-establishment of the all-powerful habit and expectation on which they rest. Mechanical and medical means may be used to further this design, but the routine employment of drugs without this end in view does but rivet afresh the assumption and habit of failure. That part of our conscious life to which, after the early years of childhood, the control of such functions as these is delegated should be taught again to work in harmony with our general needs.

But the doctor is seen also in his function of educator when dealing with clinical cases of a commoner sort. Sometimes a process of training must begin afresh because the old machinery of the nervous system has become defective through disease. No year now passes without its records of new successes in this line.

Thus, in spite of all the study that has been given to the treatment of locomotor ataxia, it is only recently that we have learned [under the leadership of Frenkel] that patients with this disease, even when bed-ridden from incoördination, can be taught by systematic gymnastics to use their muscles anew, and can thus regain to a considerable degree the lost control over their limbs. A therapeutic discovery like this is a sign of promise, and an inspiration to new research in other lines. The ataxia of the muscle typifies also the ataxia of the mind and of the other functions of the nervous system. We pay dearly for the possession of the intricate machinery which gives us our vivid imagination, our retentive memory, and that power by which we are able to grasp at each moment all the threads of our past experience and weave them into a new fabric for the service of the present. Mischief begins when the demands of this service cannot be properly met, and the different parts of the vast machine run too fast or too slow; the delicate adjustments fail to respond or work too fine. Then the heart palpitates which should simply strengthen its beat to meet

a need for more blood. Then a bronchitis provokes an asthma, and this recurs and becomes a habit. Or a painful impression that should have disappeared with its cause, swept down the swiftly flowing stream of consciousness, is caught by a backwater, or rolled to and fro in an eddy from which it cannot escape. Or, again, the instinct of imitation, imitation of others or of ourselves, makes us copy and perpetuate a bad trick of muscle or of thought. And so arise the *Diseases of Morbid Association and Habit*,* the caricatures of the arrangements and coördinations of health. Here is a singer† who in the first days of his career found himself with beating heart‡ and shaking voice in consequence of a stage fright. He is now a veteran, and no longer stirred by fear of failure, yet his old fright remains indelibly stamped upon his trembling muscles, which do their best, in their turn, to drag back the old emotion. There is at first a divorce between the bodily manifestation and the mental state, and then a new union on abnormal lines.

"I am not frightened, but my body is," said an intelligent friend. "It is my body trembles at the sight of the dangers into which my triumphant soul will carry it," said a hero of an earlier day.

Or, again, to take an example of a habit disease of another sort, here is a patient who has had an inflamed knee. It is long since the local disorder passed away, yet the pains which used to attend it recur at every trifling

* At the last annual meeting of this Society various types of habit neuro-psychosis were described by Dr. Edward Wyllys Taylor.—*Boston Med. and Surg. Journal*, July 21, 1898.

† This illustration is given by Dr. Morton Prince, and I take occasion, in citing it, to call attention to the excellent work which he has done in this direction. See various papers published in the *Boston Med. and Surg. Jonrnal*, 1898; also Association Neuroses, *N. Y. Journal of Nervous and Mental Diseases*, 1891.

‡ The presence of these cardiac and circulatory phenomena, as a part of the habit neuroses, is a complication of a peculiarly distressing and important sort ("Angst-neurosis"). [See "Heart Fear," by W. T. English; *Med. News*, April 29, 1899; also L'Education de la Volonté; Payot.]

movement which even remotely resembles those that used to drag upon the injured part. The demoralization following illness, or misfortune, or accidents, is a rich source of such disorders. A patient has haunted our hospital-clinic for a long time who thirty years or more ago received a blow upon the head which set up a spectral pain that has been his constant companion ever since, though even now he looks in blooming health. Any innate weakness which prevents the brain from carrying on its normal function of prompt, efficient reaction may allow morbid tendencies of these sorts to establish themselves, as weeds grow in a soil too poor to support a better product.

It is not correct to say that these pains are "imaginary." Regarded psychologically, they are just like all other pains. On the field of battle even the ordinary physical causes of pain may fail to cause it, for the impression is followed by no reverberation within the soldier's conscious attention. In the conscious attention of the over-sensitive patient, on the other hand, this reverberation is excessive, and thus the merest hint of physical cause, a whisper of apprehension, the influence of cyclic recurrence like that which reproduces sleep, or even the influence of memory, may make the pain come back.*

Fortunately, the knowledge that so many of our ills have this mental origin stimulates to the search for new means of cure, for new means of securing some strong outward trend for the active forces of the brain and thus inducing a little of the healthy anæsthesia of the battle-field.

The patient finds it hard to credit the fact that his suffer-

* Dr. Morton Prince has suggested that the excessive tendency to the mental representation of pain shown by certain persons justifies the designation of them as "algesics," just as other persons to whom visual images readily occur are called "visuals," and so on.

Dr. Mary Putnam Jacobi thinks it possible that the increased sensitiveness of the "pain-memory" may be a part of the general increased attention to the history of the past, though a morbid example of this tendency. *New York Medical Journal, April*, 1898.

ings are due to mental representation alone, and no wonder. For by what criterion can he, unskilled and anxious, be expected to decide, when the physician, with all his means of diagnosis, is so often in doubt? Frequently, of course, the symptoms are only partly due to habit, and partly also to some local cause. Every good physician is aware of this, but it is not so well known that the removal of other physical disorders perhaps not originally connected with the symptoms at all, but now a real complication, may induce a partial or temporary alleviation, which will supply the very impulse that was needed to give new hope to the mind and a new "set" to the functions of the nervous system. Thus it is that the treatment of trivial diseases of the eyes or joints can sometimes work such wonders; and thus, too, the new doctor so often eclipses the old. On the other hand, the keen physician will not let himself be trapped into the error of concluding that he has found the cause of the disease because he has found the cure. This is a piece of false reasoning that has worked much mischief and has often brought an unmerited credit to immature theories of disease.

The subject of the *social etiology* of "habit" disease has many interesting and important aspects. Prof. William James* has recently suggested that many of the racial peculiarities, such as the high-pitched, anxious voices, the hurried manner, the "over-modelled" faces that do not easily relax, the tense and exhausting eagerness, which visitors see so often among the people of old New England, are due not so much to excess of sunlight and dry air, as to tradition and imitation. A comparative study of the varied races of which the population of New England is now composed, made from this point of view and as a basis for more rational treatment, would be heartily welcome. This or that American or Irishman or Jew cannot be treated to the best advantage so long as he is regarded

* Talks to Teachers. 1899.

solely as an individual. His racial traditions and temperament should be taken instinctively into account, just as the pianist (to borrow a simile used by Kries) on starting to play a piece of music, glances once for all at the signature which indicates the key in which the piece is written, and then, without need of specific reflection, goes on throughout playing the notes with their appropriate sharps and flats.

To learn to do this sort of thing; to speak and comprehend the mental language of all sorts and conditions of men, is one of the many important tasks which the physician will in time learn to perform.

The existence of these habit diseases is known to every doctor, but this very fact is liable to give one a false sense of confidence in his ability to recognize them. Common as we may think them, they are yet commoner than we think. The most striking forms are seen in hysteria, the chief characteristic of which is that the patient's consciousness is split up into more or less separate groups of forces, which strive with each other for the mastery. The patient is no longer a consistent unit, and is forced, against his will, to play, with the skill of genius, the rôle of disease. But hysteria is not the only soil on which these disorders grow, unless indeed we admit that the hysterical tendency is universal, and that hysterical disabilities and eccentricities are but acute forms and caricatures of the disabilities of normal or quasi-normal life. The term "hysteroid," which retains the notion that such affections are due largely to mental representation, is often an appropriate designation.

The "accident neuroses" form an especially important sub-group, the more so that one can trace, both in the influences which make certain classes of patients peculiarly susceptible to them, and in those which finally excite the outbreak and give it its special form, the play of forces that recall "hypnotic suggestion," and are seen at work on a

large scale in mob-madness or panic. The patients who suffer from the severer forms of these disorders are, strangely enough, of the mechanic and wage-earning class, who, although sturdy, and used to hardship of various sorts, are apt to be lacking in social and general experience, and are not trained in the sort of self control that Society expects from its members, while, at the same time, their lack of a fixed source of income makes a period of enforced idleness a matter of great moment to them. They are not, as a rule, predisposed by neurotic inheritance, and have usually been strong and healthy, but the traditions of the community in which they were born and bred inculcate the instinctive belief that accidents are terrible events and lead to mysterious and complex troubles. To the patient whose mind is thus stocked with traditions and instincts sympathetic to misfortune there come next the startling and disabling circumstances of the accident itself, which dethrone the self-control and profoundly disturb the emotions and through them the action of the circulation and the heart; and to this is added the special "suggestion" furnished by some local injury or special fear. It is then as if some one had whispered to the demoralized patient, "your arm will be paralyzed"; or, "you will be an invalid for years." These cases are extremely numerous and immense sums are paid on them in damages. The general practitioner can do the community great service by recognizing and spreading the view that serious as such illnesses often are it is largely mental causes that make them so. A stalwart position of hopefulness taken by the attending physician, at the outset, as a basis for a persistent and thorough treatment by encouragement, and explanation, and personal influence, before morbid habits of invalidism have become ingrained, may save many a patient from a demoralization that the money secured by the verdict cannot make good. How often, on the other hand, does the doctor say: "Madam,

you really ought to take care of yourself; you are far more severely injured than you think you are." There is usually a period, to be sure, at the very beginning of the case, when it could be said with truth that the patient is "more severely injured than he thinks he is," because the disturbance that is going on in the deep sea of his mental life has not yet reached the surface. But for him this is a blessed ignorance, and it is a misfortune when the time comes that it is exchanged for a delusion as to the real nature of his disease. It is not the hope of gain that is the prominent factor here, in spite of the raillery of the court room. Doubtless it plays its part, but the essential influence is the disorganization of the forces that rule within the mind, and that instead of acting in harmony, as they should, break into disorder, like soldiers in panic, and then instinctively seek to readjust themselves under any tolerable, if imperfect, organization and leadership. Happy it is for the patient if this reorganization is effected under the guidance of a doctor who has had the fortune to be graduated with high marks from some philosophical department as admirable as that of our good foster-mother across the river, and thus buoyed up has brought an undivided will to the study of medicine.

In the treatment of all these "habit" cases, a persistent and insistent *encouragement*, based on a physical examination of rigid thoroughness, and accompanied by an *explanation that has the force of encouragement*, and leaves no doubt as to the physician's conviction, will often clear out the weeds that block the flow of the river of consciousness and let the current sweep along, with the self-obliviousness of healthy activity, in its old channel.

It is a striking fact, as regards explanations, that they have much greater weight after the physician's authority has become established by the partial success of his treatment. The arguments offered beforehand may have been

just as convincing as those offered later; so delightfully logical, in fact, that the physician often buttons on his overcoat and leaves the patient's door with the self-satisfied conviction that the battle has been won for both parties. How great then is his surprise, amounting almost to indignation, when he discovers, at his next visit, that not even the outposts of the enemy have been won. Let the patient's confidence have been gained by a little success in treatment, however, and he may accept [sometimes too readily] any explanation, logical or illogical, that the doctor is inclined to offer. All mental healers know this well, and have known it, doubtless, since long before the day when the doubting and suspicious Naaman professed himself ready to accept the religious views of Elisha in return for the cure of his leprosy. The explanations of the doctor are in fact quite as much an appeal addressed to the faith as an argument addressed to the reason.

It will be remembered that Naaman, who was a captain of the host of the King of Syria, was afflicted, according to the diagnosis of the times, with leprosy, and that he was sent by his master to the King of Israel, at the instance of the captive Hebrew maid, to be cured by Elisha the prophet. "So Naaman came with his horses, and with his chariot, and stood at the door of the house of Elisha." Perhaps in his heart he expected failure, and expected it the more that he had pictured to himself just what steps the prophet would take to cure him. When, then, instead of coming down to meet him in person, Elisha left him standing with his retinue at the door, and sent word to him that he should go and wash in Jordan seven times and he would be clean, Naaman turned and went away in a rage, saying, "Are not Abana and Pharpar, rivers of Damascus, better than all the waters of Israel? May I not wash in them and be clean?" And it was only when his servants pointed out to him the folly of his childish passion, saying,

in effect, "If the treatment does you no good, it will do you no harm," that he consented to the prophet's conditions. Consent he did, however, and not only was he cured, but he promptly became a convert to the true faith, and one may suspect that he would have been willing to accept any proposition that Elisha had chosen to offer. This parable, as one may call it, is instructive in many respects, besides that of indicating the power which the physician acquires over his patient through his success in treatment. It illustrates strikingly with what complex feelings the patient goes—half trusting, half doubting—to see the doctor; and illustrates, too, how the doctor is sometimes put to his wits to see that he does not discount his authority with the patient by letting him dictate the details of the treatment.

I permit myself to say, however, that such an enforcement of conditions unmeaning in themselves, although it is an effective device and often a necessary one, represents a means of maintaining authority which every physician should try to eliminate, more and more, as the relations between himself and his patient become established on a more natural basis. We must cure our patients if we can, even if it is necessary to put on the manners of the Grand Pasha, or to hold ourselves as remote as the Mikado. But on the other hand something may be risked in the interests of directness and simplicity.

Where simple encouragement and explanation fail, it is often possible to induce the patient, by coaxing or training, to do something which even he must see that he could not have done if his own diagnosis had been correct. If, for example, he has lost temporarily the power to move a limb in a certain way, he can perhaps succeed, under assistance and supervision, in making a quick jerk which will include the lost movement among others; and then he can be taught to analyze the sensation and to differentiate his efforts. Even pain (in suitable cases) may gradually be

cast out if the patient is encouraged to note that "this time," or "that time" it was less severe, until he learns to have an alternative picture of himself as free from it, which at first he did not have. Fortunate it is if the patient can be led to take interest in the "habit" theory, and still more fortunate if he takes to himself the credit of the cure.

Where all these means fail the physician must make his influence strike deeper. He must himself recognize that habits persist partly because they represent the active intervention of a portion of our consciousness which lies outside the field of our personal attention; beyond the direct reach of our personal will. This hidden consciousness, that plays so active a part in causing our morbid acts, is no stranger, but on the contrary an old friend. The power that thrusts an obnoxious act or thought into the current of our lives is closely related to that power that notes the flight of time during the night, waking the nurse at the appointed hour, or to that which tosses up to us the memory of a forgotten engagement—sometimes alas too late—as a chip is tossed up by an eddy in the stream. In the technical language of the day this power is called "our subliminal consciousness," that which works "below the threshold" of our personal attention. I prefer myself to call it the "sub-personal (or extra-personal) consciousness," but it will be better if you will forget names and seek in your practical experience the warrant for the truth of what I say.

Every thinking person is aware that our minds work under many masters; we might designate them as our "silent partners." Under normal conditions we have reason to congratulate ourselves at the useful division of labor which this arrangement secures. But when the mental harmony is broken by disease, these masters of the mind jostle and trip each other in their efforts to restore the balance; and then we feel the sort of dismay, the sense of alteration of our-

selves, such as in old days was called possession by the devil. The acts that we do are only partly the acts of our personal will. Some of them seem inspired and even performed by a power which is more or less foreign, yet which we feel bound to obey. So Tennyson's sailor boy, who must and will run away to sea, says:

"My mother clings about my neck,
My sisters crying, 'stay for shame';
My father raves of death and wreck,
They are all to blame, they are all to blame.

God help me, save I take my part
Of danger on the roaring sea,
A devil rises in my heart
Far worse than any death to me."

Or, as the writer of the charming Harvard ballad puts it:

"Within my earthly temple there's a crowd;
There's one of us that's humble, one that's proud;
There's one that's broken-hearted at his sins,
And one that, unrepentant, sits and grins;
There's one that loves his neighbor as himself,
And one that cares for naught but fame and pelf.
From much coroding care should I be free
If once I could determine which is me."*

We have not the right, even where we have the wish, to deny our responsibility for the acts of these hidden forces of the mind, and it is a high function of education to enable us to understand and to control them; to make the desirable "me" prevail.

And thus queerly constructed, and perhaps with the demon of distrust looking through his eyes and speaking a language which his words vainly strive to contradict, the patient comes to the doctor, carrying his burden of morbid habits, as Christian in Pilgrim's Progress carried the burden of his sins, seeking for explanation and relief. How shall he be met?

The answer instinctively suggests itself, by some sort

* E. S. Martin, Sly Ballads in Harvard China.

of treatment akin to "hypnotic suggestion." This name excites great prejudice in many minds, and I should be glad to have it forgotten until the principles on which such treatment rests shall have been indicated, and the fact made evident that the methods designated differ only in degree from other modes of influence such as every physician uses in his daily practice and every person of strong character in the contacts of daily life. The analogies that suggest themselves in these directions are, in fact, so striking that there is risk of going too far toward making out the hypnotic method to be an affair of everyday occurrence. One often hears the influence which society exerts over its members spoken of as "hypnotic," and it is quite as justifiable, and more to our purpose, to extend the conception so as to cover the case of a hypochondriacal patient whose judgment and reason have been captured by a morbid habit or a delusion of disease. There are times when we all become unusually impressionable to external influences or to the promptings of our own instincts; and it is clear that this is made possible by the withdrawal of the critical and questioning faculty that stands like a Cerberus at the door of the mind and is the enemy of all impressionability except of its own choosing. It is by no means to be assumed that the person thus altered is necessarily reduced to a lower plane. We make ourselves voluntarily more impressionable and less critical for the purpose of listening better to the whisper of our genius or our conscience, or to the whisper of one such voice chosen out of many:

> "Though love repine and reason chafe
> There came a voice without reply,—
> 'Tis man's perdition to be safe,
> When for the truth he ought to die.' "

Even the means by which the impressionability of health is voluntarily secured sometimes present a striking resemblance in detail to those which are used for the purposes of

hypnotic suggestion. Take, for instance, the case of a person who is striving to recall some name or fact. The whole substance of his effort consists in eliminating everything that stands in the way of complete passivity. He sits fixedly gazing at some object, yet taking no heed of what he sees; he hears the ticking of the clock with unusual intensity, but the sound does not reach beyond the ear, and serves only as a means to keep him from entertaining any stronger interest that might prevent his catching the first glimpse of the real object of his desire, if, by chance, impelled by the hidden forces to whose influence he submits himself, it should drift across the field of his mental vision.

I need not pursue these analogies further in order to convince you that a patient staggering under a tormenting and dominating habit has no reason to feel humiliated when he yields to an influence so familiar as that which I have indicated, in order to make himself impressionable to the words of encouragement from the physician in whom he has confidence, and who is striving by these means to reveal to him another and truer picture of himself than that with which in his delusion he has been so long familiar. And this applies also to the child, impressionable by nature both to evil and to good, and sometimes a victim to tricks of bodily and mental habit which can be removed only by deep reaching influences, if even then.

A sweeping condemnation is often brought against the use of hypnotic suggestion, based on the assumption that it involves the subjection of the will of the patient to that of the doctor. But this same sort of influence is exerted daily, in the schoolroom, in social intercourse, and most of all in the army; and the results are good or bad according to the obvious intention of the method, and to the intelligence or ignorance, the high-mindedness or low-mindedness of the person by whom it is brought to bear. The physi-

cian who domineers over his patient or makes him a slave to needless rules, can weaken his independence without hypnotic suggestion; while he who is bent on strengthening the patient's will can and must call special methods, analogous to those of hypnotic suggestion, to his aid. Nevertheless the final aim of the doctor, as Madame De Stäel said of the legislator, should be to make himself unnecessary; and we may add, in company with the sturdy Sir James Paget, to make, if it is warrantable, even his rules unnecessary.

For the attainment of the ends that I have indicated, the patient must be taught to let his critical instinct and his will lie passive at first, while the new set of motive-impulses is being presented, but finally to use them for reenforcing the new mental attitude which they could not have created alone. The part thus assigned to the will is not a wholly negative one, and the imputation that the patient grows strong solely through another's effort is only correct if the treatment stops short of the final stage. In fact, the whole treatment, from first to last, and at every stage, involves the patient's consent and coöperation. This passivity or "consent," whichever one may call it, of the critical instinct and the will may be induced in many ways. The teacher secures it when he exerts over his pupil the fascination of his personal influence, for the purpose of interesting him in his tasks; the physician secures it when he induces his patient to sit or lie quietly with muscles relaxed and senses engrossed, so that he shall listen without inclination to comment on what is said, or shall allow his own sense of encouragement and hopefulness to take deeper root. If, after such a treatment, the patient comes and says: "I heard what you said, but cannot now repeat it," and if, thereafter, his acts show the re-appearance of the newly-suggested impulse in the form of a spontaneous product of the patient's thought, the most desirable result will

have been attained. This kind of treatment may be called "hypnotic suggestion," if one will, but the prejudices that this word used to excite would never have arisen if such therapeutic measures as these had alone been in question.

It would, however, be disloyal not to recognize that even the experimental uses of hypnotic suggestion, objectionable as in some respects they are, have been of high value to medicine. It is to the studies made possible by hypnotic methods that we owe much our of present familiarity with that part of the mental life that lies beyond the direct reach of our personal attention, and if to this we add the service that treatment involving profound hypnotic influence has rendered in many cases of serious and otherwise incurable disorders we have abundant reason to be grateful for Braid's and Liébault's discoveries. Nevertheless, it has, fortunately, been found that the actual induction of the hypnotic sleep is far less often necessary, so far as therapeutic purposes are concerned, than was formerly believed. The results effected through it have given the hint that simpler methods might suffice, and so have inspired with new power the physician's confident assurance of improvement or recovery that gets the name of "waking suggestion." It is the patient's misfortune if this assurance does not reflect the most thorough diagnosis that science can command and the most hopeful prognosis that the conditions, *taken at their best*, can justify. Unfortunately, it sometimes happens that the very scientific training which justifies the physician's dignified position of authority in the community acts to prevent him from "taking at their best" the influences that make for a good prognosis. The capacity, one might almost say the genius, for improvement which the patient often shows is apt to be underrated, just because it is so mysterious and so undefinable. Both physician and patient need to realize more fully that the latter has the germs of a real power of control over his bodily and mental

states, provided his personality is conceived of in a sufficiently broad sense.

Whatever be the method by which the influence is secured, the treatment of these diseases of association and habit must follow one of two familiar courses. The first is that of eliminating from the mind the troublesome and recurrent impulse or habit; the second, that of introducing another impulse incompatible with the former and tending therefore to its exclusion. Both of these attempts are made in education of every sort, but it is safe to say that the latter is by far the more successful of the two, and is in fact the one which mainly does the work even when it is not ostensibly employed. It is better to build up than to pull down, to induce virtue than to eliminate vice, to supplant morbid thoughts by others that are better, or to utilize them as materials for growth rather than to conduct a campaign against them. The restoration of motion is the essential factor. The swiftly-flowing river will quickly absorb the muddy brooks that enter at its sides. That is, to say the least, the "Western" as distinguished from the "Oriental" mode of progress. It is true that great therapeutic advantages have seemed from time to time to have been gained by seeking out and casting out a painful or bitter experience which had been germinating, perhaps for years, in the mind, hidden from view except in its morbid manifestations, which took the form of hysterical outbreaks, moods of depression, disordered sleep, and other phenomena of like character. Several remarkable series of cases* have been reported where such ideas have apparently been traced to their lair and then exterminated, and with them the complex organization of morbid ideas and emotions of which they formed the centre. I believe, however, that this explanation of the

* See contributions by Dr. Russell Sturgis on the Use of Hypnotism to the First Degree, in the *New York Record*, Feb. 17, 1894; also *ibid* 1899. The earlier publications by Janet, Breuer and Freud, and others should be noted in this connection, and also Loewenfeld's excellent criticism in his Psychotherapie.

therapeutic successes which have unquestionably been achieved in such cases is not necessarily the correct one. One may readily believe that if a patient could be brought to give his tacit consent to any theory of the cause of his troubles which the ingenuity of the physician might suggest, he would gain new hope and receive an effective impulse toward recovery even if the theory which was offered him was not the true one. The real point is, I believe, to secure a nucleus for a new set of associations which shall be exclusive of the old ones. The patient is to be taught to push ahead in the direction of health and activity as one pushes his way through a thick forest, regardless of the scratches and bruises to which he is exposed on the way, and it does not greatly matter whether the path of escape is or is not identical with that which was followed when the way was first lost, except in so far as that it is important to gain the patient's ready acceptance of the path actually proposed.

Unfortunately, even the much vaunted hypnotic suggestion often fails to work; or if the new suggestion seems at first successful the morbid habit may in the end settle heavily back again. Is "Philip drunk" or "Philip sober" really the stronger and more consistent character? This is a question which the doctor often has to ask himself and to which he can perhaps find the answer only by trial. Fortunately, in the memory of Philip sober there may be groups of half-forgotten experiences* dating back perhaps to the days of his youth, and capable of forming, by their union with the new impulse, a temperament sympathetic with the better course, and thus he may be enabled to prevail. One may try to express such conditions in terms of cerebral anatomy and physiology, but it is doubtful whether, in the present state of our knowledge, a clearer conception of the process would thereby be gained.

* See Dr. Mary Putnam Jacobi's paper, "A Suggestion in regard to Suggestive Therapeutics," in the *New York Medical Journal* for April 9, 1898.

Fortunately for the educational outlook, the evidence has begun to accumulate that a morbid inheritance is not the inevitably crushing and baneful thing that it has been thought. We come into the world, each one a being of limited capacity but in other respects free to become what circumstances make us, and responsible, to the extent of our capacity, for our lot. We bring no ticket-of-leave which stamps us as drunkards or maniacs on probation, but we do bear, in the histories of our ancestors, a certificate that hints by what efforts and by what avoidances we can make ourselves reasonable successes in our respective lines. There is no original sin, and not even, as it seems to me, original propensity, but only original capacity and original limitation, and even limitation is only another name for latent capacity.

A highly interesting side-light is thrown on the discussion between these two doctrines—the fatalistic doctrine of propensity and predestination, and the more hopeful doctrine of plasticity limited only through capacity—by the evidence adduced in a somewhat similar discussion which has been going on among the embryologists. What is it that gives our bodies their definite and ever recurrent form? Do the nerves as they pass from their centres of growth to their destination in the skin and in the muscles bend and twine to find their passage in obedience to some inherent tendency of growth, or does each cell-process start out not only ignorant of its future but without a positively defined future, and make its way in obedience to mechanical laws of least resistance and simple laws of growth just as a rootlet makes its way amongst the stones, in its seemingly inspired search for the distant water? If I understand aright the teaching of the present day, the latter is the case. This teaching does not aim to rob heredity of its powers, but, in so far as it is applicable to the problems of mental growth, it does glorify the influence of education in making us what we

are. The child of the drunkard does not inherit drunkenness, but he does inherit a defective brain; one liable to go wrong perhaps in many ways but reclaimable to a considerable extent through education; just as the child of the tuberculous parent does not inherit tuberculosis but the soil on which it may develop. The difference between these two doctrines is more than an academic quibble. It is one of vital tendency.

The cases that I have thus far had in mind have been of relatively curable sorts, either because the patients had traditions of good health to look back upon, or because it was assumed to be possible to cast out some special fixed idea, or habit, or delusion of illness, which had arisen in consequence of some more or less accidental experience but still lay loosely attached in the mind, or, at any rate, had not become, through the bonds of association, an integral part of the great body of ideas and feelings on which the patient's mental life revolved. But these disorders arise more readily, strike their roots wider and deeper, and keep a more tenacious hold if there is a native defect in the mechanism of the nervous system such as we see in that great class of cases which for convenience's sake may be grouped as *constitutional neurasthenias*. With such patients the problem of treatment is often not so much to rid them wholly of the consciousness of their symptoms as to aid them to look through and beyond their troubles, and eventually in some degree to ignore them, as one learns to ignore a defect in vision. If patients with this sort of illness are often plagues to themselves they are no less a source of sighs and groans to their doctors and their friends. Yet they are among the best and keenest people in every community, and it may even be said that it is their doctors and friends who are in a measure responsible for keeping them what they are—at least in the sense that salt was defined by the small boy as that which made potatoes taste so bad when there wasn't

any on. In other words, they would be better off if we knew better how to appreciate and to treat them. Liable to fatigue, and unable to accomplish as much as their robuster neighbors, such patients become keenly alive to their failures, and are apt to fall into mental habits marked by baffled reason, morbid self consciousness, and balking will. Beginning by suspecting and hesitating, they often end by ceasing to act. The limitations set by the original "weakness" are of trifling consequence as compared with these secondary and needless incrustations, framed from false premises by the patients' inexorable logic. But the balking will like the balking horse can often be trained to work again; the obstacles that seemed so invincible when tangled into a web made up of the memory of past failures and fears can often be overcome if taken piece by piece.

Fortunately, it is by no means every neurasthenic patient whose case takes the unfavorable course that I have indicated. There are many persons, weak in body but unconquerable in spirit, who keep themselves free from this entangling web of troubles. They make unnecessary the therapeutic suggestions of the physician by accepting their limitations as indications for casting about to decide on their best course for the future and best means of usefulness for the present, and striving to regard the tasks imposed by illness in the same light as the tasks imposed by health. Such persons give new meaning to the fine words that Tennyson puts into the mouth of the aged Ulysses:

> "Tho' much is taken, much abides; and tho'
> We are not now that strength which in old days
> Moved earth and heaven; that which we are, we are;
> One equal temper of heroic hearts,
> Made weak by time and fate, but strong in will
> To strive, to seek, to find, and not to yield."

The problem for the physician in dealing with patients to whom such a course as this does not come instinctively is

to use all the means that may lead to its adoption. If there is any slender thread of interest or enthusiasm or even of fanaticism in their composition it should be recognized and encouraged. And, happily, the slender thread is often enough. It has not the all embracing power of the large and warm temperament of hopefulness, but what is lacking in massiveness may in a measure be made up for by intensity. The almost fierce glow of energy and fortitude seen in some of the frail-bodied New England women portrayed by Miss Wilkins gives a good illustration of this principle.

Neurasthenic patients cannot, as a rule, be expected to overcome their troubles alone. We say to them, "make an effort of the will and do this or that," but the will cannot create situations but only choose between them, and although it is true that a better choice usually exists than that which the patient makes, yet to see this and to choose correctly is a task of no little difficulty. Such a patient is compelled at each step to compare the act which he wishes to accomplish with that which he wishes to avoid, and so the latter is presented afresh with all its imperative fascination, as a card is forced on a novice by a skilful player.

It is the physician's part to present a new alternative for the reason to adopt and the will to act upon. When such patients clearly understand that with patience and the cultivation of a dogged indifference to symptoms which are unimportant though annoying, they can learn to note and to accentuate their powers of accomplishment and enjoyment, the ground is already cleared for the action of food and tonics, recreation and active work.

An element of special encouragement and mental stimulation in particular directions may be made to accompany treatments designed ostensibly to fulfil other needs. The famous so-called "rest cure," which the genius of Weir Mitchell devised, owes the remarkable success which it

exhibits in able hands less to rest and food in themselves than to the mental influences that go with the isolation, the scrupulous precautions that leave no excuse for indigestion or fatigue, the obvious confidence of the doctor and the nurse, and the contagion of recovery such as permeates every successful hospital. All these changes in the patient's surroundings are often, from the point of view of mental influence, more effective than a journey to Europe, though even as regards the latter the familiar lines:

> "Coelum non animam mutant,
> Qui trans mare currunt."
> (They change their skies but not themselves,
> That cross the seas)

are, luckily, not always true.

Many a doctor has tried the rest treatment and has failed, because he did not follow exactly Dr. Mitchell's rules, not realizing that in omitting certain "surely unessential" details he was leaving out the very heart of the mystery. On the other hand, I fully agree with Dr. Prince* that results similar to those accomplished through the agency of the "rest cure" may often be obtained through pure mental stimulation and education.

Take next the question of physical exercise, which may stand as a type of a whole class of treatments. You recommend it for your neurasthenic patient, but to what end? Do you wish to toughen his muscles, to help his digestion, to strengthen his heart? Or is it in fact recommended for the sake of introducing the leaven of energy, in the hope that it may finally infect his whole mental life? If such is your desire, five minutes of sharp exhilarating work or play will do what hours of chest-weights would fail to accomplish. Perhaps you may even start a love for the study of physical development and thus give a new impulse to an invalid life. It may be as when a boy, dull at his

* Educational Treatment of Neurasthenia. *Bost. Med. & Surg. Jr.*, 1898.

books and drifting into general incompetency, has the good fortune to meet some one who discovers that he is a genius at whittling, and who by encouraging him in this accomplishment gives him a chance which will in the end transform him not only into a good mechanic but into a man of first-rate, all-round capacity. And so, too,* relaxation of body may be employed to teach relaxation of mind. On the same principle, a soothing treatment by water or electricity may help to give the genius of sleep a better chance to control the currents of the mind.

It is worth while to note what a needless weight of sombre forebodings is often carried by a *word* or *name*, to the patient's anxious mind. To feel "tired" may be bad enough at the best, but it makes a world of difference whether one accustoms himself to take the term as meaning the fatigue of a person who expects in due time to be rested, or as a bottomless pit of exhaustion, demanding sighs and groans. If it is true, as Dr. James says,† that the drunkard's reform often dates from the day that he gives himself the unsavory name, it is equally true that needlessly alarming symptoms can often be unmasked on the reversed principle; as a legal friend of the writer once, in court, robbed an "excoriated tibia" of its terrors by rendering it as "barked shin".

Happy is the neurasthenic patient who has a sense of humor or a liking for philosophy, so that he can get a certain satisfaction from externalizing his troubles, as William Blake painted his visions and Bunyan put his into the Valley of the Shadow of Death. To learn to recall the good days or the bad days; to remember that the University of Leyden was founded during the starvation of the great siege; and to realize that limitations are also finger-posts to new sorts of progress, infuses a sense of triumph into one's efforts toward the self-forgetfulness of effective life.

* As Miss Annie Payson Call has so well explained.
† Talks to Teachers.

Many patients think that they are not gaining, because the signs of improvement are less obvious to them than the signs of failure. The latter stand out in painful isolation; the former lose their own identity in augmenting the general efficiency, just as the stippled spots of paint on the impressionist's landscape blend into a glow of color. Fortunately, however, the test of successful work and the sense of comfort that accompanies it usually tell the truer story, and the physician can powerfully intervene as an interpreter.

Neurasthenic patients are often led, partly by native temperament, partly through the circumstances of their illness, to seek philosophic views of life, and indeed without some familiarity with them even the buoyant satisfaction that successful work brings to the healthy mind leaves something to be desired.

The inspiring doctrine of individuality, with which the pages of Emerson are full, that heightens one's interest in the novelties which the light coming through one's own window, the touchstone of one's own genius can alone reveal; the reassuring belief that urges us to "live ever in a new day," and forbids us to drag around the "corpse of our memories" of evil; the great doctrine of "service," which presents only another aspect of the principle that if a man consistently follows the lead of his own best instincts he is sure to find that his interests are at one with those of an ever-widening community; such principles as these are like so many watch-fires, blazing up from the hill-tops into the darkness of the night.

In conclusion, I would say, as I said in opening, that every physician is an educator, whether he will or no, and should study to be a good one. He works of necessity with a threefold object in view; to cure his patient; to leave him better able to cure himself another time; and to establish systems of treatment that shall reflect sound and liberal views. He must of necessity come into close per-

sonal contact with his patient, and may, therefore, through the powerful influence of his own personality, make him narrow, or suspicious, or unduly dependent on rules and mysteries; or, on the other hand, more reasonable, more self-reliant, more liberal, a firmer believer in his own genius, and the supporter of simpler creeds. The opportunity for usefulness is an enormous one; for I hold that to rid a patient of a tormenting delusion, and to increase his power of resistance against debasing habits and thoughts, is quite the equivalent of a successful operation for a painful disease, and needs as much skill and preparation. It is to the opportunities and responsibilities of this task, which should not be evaded, that I have tried to call your attention.

The physician too must call the community to his aid. For many of these disorders start in the imitation of inferior social customs and in the acceptance of narrow social traditions, and we may confidently hope that they can be eliminated in part through changes in public feeling such as an intelligent doctor may help to set on foot. The community is neither intentionally ignorant or intentionally heartless. It adopts the easiest and nearest opinion and is readily open to change. We gather at our annual meeting to gain strength from the contact with each other, as Antæus from his Mother Earth; let us separate again, each to be a centre for the spread of knowledge not only of those forces whose effects are to be measured by the microscope and the chemist's tests, but of those also which no chemist can estimate, but which may, nevertheless, work upheavals in social sentiment, as the water which trickles into the crevices of the rock may, one day, freeze and burst it asunder.*

* I am aware that an account of "mental therapeutics" could make no claim to completeness, even in outline, if it did not discuss the limits of action of this agency. Nevertheless, this part of the subject is too large to handle briefly, and too important to dismiss summarily. I therefore leave it for another occasion, and content myself with a reference to the treatment of those affections which the general practitioner must treat on the lines here indicated, if he wishes for success.

ARTICLE III.

# IMPROVEMENTS IN ARMY MEDICAL SERVICE, SUGGESTED BY EXPERIENCE IN THE LATE WAR.

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READ JUNE 14, 1899.

# IMPROVEMENTS IN ARMY MEDICAL SERVICE, SUGGESTED BY EXPERIENCE IN THE LATE WAR.

THE object of this paper is not to criticise the medical service of the regular army or National Guard, especially of our own State, whose medical service at the commencement of hostilities was almost all that could be desired, considering that the volunteer militia had not seen active service for over thirty-five years. It is only by experience that we can approach perfection in any branch, and my idea is to suggest methods for improvement in our service.

As surgeon in charge of a large field hospital, I have had exceptional opportunities to observe defects in our present system. No matter what advancements are made in National Guard medical service, one cannot fully utilize them in National as in State service, for the reason that in the former we are depending on the efficiency of superiors over whom we have no control; but the State training of its medical officers in a body and the individual training cannot fail to produce good results. To illustrate this point: The country has another war; Massachusetts sends to the front, as it recently did, medical officers well trained in the duties of their department; each regiment, as in this war, is sent to a brigade composed of troops from different States, whose medical department may be controlled by an officer of little experience in caring for large bodies of troops. Necessarily, the inefficiency of the head

of the department must hamper the work of our medical officers; for instance, take the matter of supplies; if the superior officer does not furnish the proper medical supplies, the command must suffer; but with subordinates who thoroughly understand red-tape, the career of the inefficient will be anything but pleasant. Then again the surgeon is apt to be detailed from his organization for special duty, but wherever he goes his individual training will help him.

What excuse have I to offer for bringing this subject before our Society? I have no apology to make; it is the proper place. Our profession is responsible, to a certain extent, for the men it sends to the medical department. For the work of our physicians on the field, the profession at large share with the military authorities the criticism which may be just or unjust, and it seems to me highly proper to interest the profession in the matter. The material, medical, surgical and hygienic, procured in war, should be placed in the hands of the best professional talent, in order to reap what profit there is to science and for the benefit of mankind. For the information of those not familiar with military organization, I will state that medical officers in an army corps (containing about 25,000 men) were distributed about as follows: one chief surgeon of corps, with the rank of lieut. colonel; two division surgeons, with rank of major; six brigade surgeons, with rank of major, one for each brigade; nine regimental surgeons, with rank of major, one for each regiment; eighteen assistant surgeons, with rank of first lieutenant, two for each regiment; one assistant was detailed for special work at division hospital, or other duties.

So many details were made from medical officers for special duty, that most organizations were handicapped by being short of physicians. Brigade surgeons existed only in name. They were detailed to take charge of division hos-

pitals and other special duty. The senior regimental surgeon was acting brigade surgeon. Contract surgeons were detailed to take the place of these officers in many cases. Many of these contract surgeons were first-class men, impelled to offer their services through patriotic motives, and I shall always recall with pleasure the able and self-sacrificing devotion of many to their duty. These men were not examined for their positions, and, consequently, some got into the service who had nothing to recommend them but their political influence. For obvious reasons every surgeon ought to be commissioned in the service, not for any vain glory, but in order to render all amenable to the same discipline, and to stimulate pride. I cannot see the reason why men should serve in this department without commissions any more than in any other. A reserve staff could be formed of physicians who came with good professional recommendation and passed an examination in the different branches of the science. If I can judge by the eagerness of my medical neighbors to go to the front, I think the flower of the younger members of the profession could be secured for this honorable and important work. When a medical officer was detailed for special work, as for instance where one considered an expert in yellow fever was hurried to the front, a member of this reserve staff, who had shown his qualifications for the work, should be detailed to take his place. He could assume his new duties feeling that he would be respected by officers and men, holding the commission which entitled him to it. Of course, it is only fair to state that the apparent urgency was so great, and the heads of departments so busy, that to make provision for this method was perhaps not deemed expedient.

Had our troops in this Commonwealth gone to the front in brigades which had long been organized, their service would have been even more efficient than it was, with medical and every other department properly equipped and an

ambulance corps that has been a pride to our soldiers. Our leaders in other departments, of the militia service as well as of the medical, are, and always were, men of ability and action. As intimated in an early part of this paper, surgeons in their respective States may have received excellent training, but in the field may be handicapped by an officer whose experience has been confined to a small military post; much of the executive ability of the former is lost by this, but even under these circumstances their training renders their services valuable, although not so much so as if they performed duty with their own organizations.

You are familiar with the noble work of the Massachusetts Volunteer Aid Association. The surgeon superintendent of their ship "Bay State" was formerly Surgeon General of this State, and his successful management suggests the feasibility of calling to our aid in emergencies the services of physicians who have served the State, in its medical department, but who have retired after nine years of continuous service, or another class that have severed its connection with the militia for various reasons. In looking over the Adjt. General's Reports of this State for the past ten years, I find the names of at least twenty surgeons who have left the service. Most of them would be proud to give their services if asked, and I can answer personally for several, who have told me that they would have served if they could have procured an appointment.

I recommend that in the future, a reserve staff be formed of all medical officers who have served in the militia. These men have received some training in the duties of the medical department and if not required in the active work of a campaign, their advice and relief work at home would be of great value. The skeleton of a first class Volunteer Aid Association could be formed by these officers, controlled by the Surgeon General, and its official connection with the service would be of decided advantage. A military organ-

ization should be able to take care of itself in every department.

At least one surgeon in each brigade, and better if in each regiment, should be selected for his skill in surgery. You can see the importance of having a trained surgeon, a specialist in surgery, in an organization that may be engaged in battle where major operations requiring great skill may be performed. The camp of instruction is not the place to teach surgery, neither is the battlefield the place to learn. In the school suggested, men will be taught surgery, particularly that which relates to gun-shot wounds and other injuries likely to be met with on the battlefield, but this instruction cannot make them skilled surgeons; a special aptitude is required, and we should have no difficulty in getting at least one first class surgeon in every organization. A special surgical examination on admission could be given to applicants seeking those positions.

A bacteriologist could be attached to each brigade or division. The Reserve Corps, before alluded to, could furnish specialists in this line. With thoroughly equipped laboratories, they could be of inestimable value in aiding the surgeons to diagnose doubtful cases of malaria and other diseases, besides furnishing valuable aid in compiling the medical history of an army, and adding to science. A Division Hospital, as its name indicates, is a hospital for all the sick soldiers of the division, that is for those ill enough to be confined to bed. In this campaign, regimental hospitals were practically abolished and all the hospital equipments called in, at least in one southern camp, to form the Division Hospital. This met with such vigorous and just opposition from the volunteer surgeons, that a compromise was made and each regiment allowed to retain two cots in its hospital for emergency and observation cases. Towards the middle of September the wisdom of allowing regimental hospitals was clearly seen and provision made for the care

of soldiers in their own regiments. I think most volunteer surgeons will bear me out in the statement that Division Hospitals are failures in the broad sense. There should be a Division Hospital for special cases; where major operations could be performed and where cases requiring the treatment of a specialist could be referred; also a contagious hospital. The regimental surgeons should treat their own men; their sick should be nursed by their own comrades; the question of discharge for disability and sick furlough should be settled by the regimental surgeon. The surgeon would not only be responsible to his superiors for the care of his sick, but he would also be responsible to his State, and his neighbors. With Division Hospitals the regimental surgeon has but little responsibility; he becomes a mere transferring agent for Division Hospitals; an admitting physician, so to speak; his men lose confidence in him. In the future, if Division Hospitals exist, they should be thoroughly equipped with everything necessary to form a first class field hospital, and not depend on the equipments of the State to furnish them. I do not wish these remarks in regard to Division Hospitals to be considered a reflection on any individual for their introduction in this campaign, for it did seem before and early in the war that the system had much to recommend it; but I base my opinion on my own observations, and those of many surgeons. However, a longer trial of these institutions may convince us of their superiority. The failure to supply these hospitals with suitable equipment is explained by the fact that the demand for medical and hospital supplies for such a large army was so great, that it was impossible to furnish them at such short notice.

In the National Guard we have not the organization for a Division Hospital, but it would be well to practise more for the perfection of Brigade Hospitals, which really are Division Hospitals on a small scale, in order to perfect ourselves in the working of larger field hospitals. We had

so little sickness during our short encampment in this State, that we had but few cases for our brigade hospital at South Framingham.

The encampment, care of men, etc., should be the same as in actual war; the drinking water *should be filtered, no matter how pure*, in order to accustom the men to it, and to impress on their minds the importance of such a procedure. A garbage plant is important; a regiment should be so accustomed to active service methods that it could step gracefully from the militia into the national service. To be sure, there would be more or less recruits in every organization at the commencement of hostilities, but the skeleton of the perfect machine would be there and would quickly adapt itself to campaign work. The soldier cannot be produced in a month any more than the mechanic.

How much have we improved our medical department since the civil war? And in what ways shall we improve it in the future? As far as the volunteer service is concerned, we have made a decided advance. Much interest has been taken of late years in the progress of this work. Education of medical officers co-incident with general medical education has contributed not a little. Antisepsis has contributed greatly, but of course we cannot claim all improvements, such as antisepsis, for the medical department; but we can claim it for our profession, and after all what we claim for our department is simply a claim for our profession, for there is no title we are so proud of as plain doctor.

In 1892, the Association of Military Surgeons of the National Guard was formed, and in 1894 the scope of the organization was enlarged by taking in the medical officers of the U. S. Army, Navy and Marine Hospital Corps, and the name changed to Association of Military Surgeons of The United States. This association meets annually, and many valuable papers on all subjects pertaining to military

medicine are read and discussed. The convention lasts three days, and the exchange of ideas and the interest awakened in subjects have done much to advance the medical service of the country.

I do not wish to take all the credit for suggestions here made. Some are original, and many are obtained from my associates in the militia, particularly the superiors with whom I have had the good fortune to serve.

A few quotations from the Surgeon Generals' reports will show the interest taken in our State medical service. Many of the suggestions have been acted upon and have borne good fruit. I trust that ere another year has passed, our volunteer militia will have profited by the introduction of all these improvements.

Surgeon General Kittredge, in 1893, recommended a physical examination for recruits, that a gymnasium be established in every armory, and a systematic course of athletic exercises be prescribed and carried out under a proper instructor, and also that a system of feeding troops in conformity to military usages be adopted, together with an emergency ration.

Surgeon General Burrell's Report, 1894:—"In a few of the States the medical department of the National Guard has been placed on a permanent footing by being made a staff corps. In one State, when a vacancy occurs in the medical department it is announced in the public press that a competitive examination will be held for such appointment, and the best man is commissioned as an assistant surgeon, and is sure of promotion and full surgeonship and possibly a directorship. Whether it were wise or not to bring about this condition of affairs in our own State, has been a matter of serious consideration; but our present system and the personnel of the medical department of this State are in such condition that it has not seemed wise to me to seek such a change, at least at present." (Some of the duties of medical officers M. V. M.)

Recommendations by Gen. Burrell, Surgeon General's Report, 1894:—"That a physical examination of recruits be made mandatory; that the surgeons receive compensation for the examination." Dr. Burrell and his successors have worked hard for the attainment of this, and now the examination of recruits is mandatory and the surgeons will receive moderate compensation for their services. Since the plan was started by Dr. Burrell our recruits have been examined to some extent through the generosity and unselfishness of our medical officers, who have worked zealously for the State without pecuniary reward, but in the future this examination will be done in the thorough and efficient manner such important work deserves, and the surgeons receive some compensation.

In the late war many officers and men enlisted who were indebted to their States for their military training, but being rejected on physical examination, were consequently unable to make any return for this training.

The war has taught the lesson anticipated by Gen. Burrell and his successors, and in the future our militia men will be ready physically as well as patriotically when their country calls for them.

Surg. Gen. Burrell's Report, 1894:—"I am convinced, from personal investigation and from reports of our medical officers, especially that submitted by Lieut. C. W. Galloupe, that a large saving in illness, even of a temporary character, might be made at the encampments of State troops by the State providing suitable food. I would by no means recommend an army ration,—not but what an army ration in itself is an excellent bill of fare, but it is not adapted to State troops who go into camp for a short time. Troops should be provided with plain, wholesome food, and this should be furnished by the State."

In 1894, Gen. Burrell adopted system of blanks modelled after those in use in the army, but so modified as to meet the wants of the State service.

A school for medical officers was started during the régime of Surgeon General Burrell and is still in existence; one or two sessions have been held every year, and many interesting papers on subjects of military medicine and surgery have been contributed. Dr. Burrell obtained permission from the Surgeon General of the United States for members of our organization to attend the school for regular army surgeons in Washington, but for obvious reasons advantage was not taken of this privilege. The starting of our school by Dr. Burrell was a step in the right direction, and the continuation of this idea on a larger scale desirable, but the formation of a special school for militia medical officers, and conducted by medical officers, is impracticable, and I would therefore recommend that a professorship of military medicine be established in the different reputable medical colleges of the United States. It seems to me that this would be a great step towards approaching perfection in military medicine and surgery. The different branches of these subjects, including hygiene, transportation of the sick and injured, the management and organization of field hospitals, red tape of the service, etc., could be taught in a course of lectures extending over one year.

Even though the knowledge thus gained were never used in army service, it would be no burden and would prove useful in civil practice. The knowledge of red tape acquired would give the student an insight into methods which would be of some benefit to him in conducting his ordinary business affairs. When the tocsin of war sounded again it should find the medical profession, as a body, ready to tender its best assistance, without the preliminary training which is now required in order to do effective work.

In the late war, our profession as a whole did noble and efficient work, and whatever may have been lacking in its effectiveness was not due to ignorance or incapacity as far as ordinary professional duties were concerned, but to inex-

perience in military medicine. It has been suggested that the national government establish a medical school for army surgeons including militia surgeons, to train physicians for the army. This is unnecessary, as the course now given in Washington to regular army surgeons, before they enter their career, is sufficient. In any great emergency, such as the civil war and the one through which we have just passed, volunteer surgeons must be depended upon for their share; they cannot devote the time to take a course at some special school remote from their homes, and the method I advocate would be in conjunction with their regular medical training. The advantages of this system are obvious. The country would then have a large body of doctors fitted for the medical service, ready at short notice. Until such a system is inaugurated, in fact well established, there should be a course given to medical officers of the National Guard in medical schools or post graduate courses, the latter consisting of at least thirty-six exercises in the form of lectures, or one a week for the school year. A school like Harvard could give one or two exercises a week in its regular course, or the State could provide for instruction in post graduate schools. This system would give an opportunity to medical officers to derive decided benefit from connection with the militia. At present the advantages are not professional but almost purely social, excepting perhaps the slight experience derived from practice at State camps. By strict entrance examinations, embracing general medicine and surgery, the Mass. Volunteer Militia has maintained a high standard in its medical department, and it is the hope of the writer to see the whole service of the National Guard brought to the same or even higher degree of efficiency, by suggestions recommended in this paper.

I quote from the late Surgeon General Forster: "Voluntary examinations of recruits are maintained in some commands; to secure the greatest efficiency of our State troops,

such examinations should be mandatory with all, and I so recommend." To establish company bearers as part of the militia system of the State, he requested the following paragraph to be incorporated in general order No. 6 :—

"Company commanders with the concurrence of the senior medical officer of the command, will designate four privates for instruction as litter bearers. They will be known as 'company bearers' and shall be instructed under the supervision of the medical officer (senior) at least one-half hour daily while in camp and at such other time during the year as the commanding officer may deem advisable, in the duties of litter bearer and the methods of rendering first aid to the sick and wounded. This special instruction will not relieve them from the performance of all regular (their) military duties. They will give first aid to the sick or wounded or carry them to the rear until relieved by members of the Ambulance Corps or a medical officer. When so relieved they will immediately join their companies.

"When on duty where their services may be required, they will wear a distinguishing mark—a red brassard around the left arm, just above the elbow."

Our present Surgeon General has well sustained the record of his predecessors. His earnestness and zeal in seeing that his medical officers were well supplied with everything needed in their department will always be kindly remembered. His motto, "There is nothing too good for Mass. soldiers" has been lived up to in the present campaign.

In his recommendations this year Surg. Gen. Blood says, "I would recommend that each Mass. Regt. of Infantry M. V. M. have one surgeon with rank of major, and two assistant surgeons with rank of first lieutenant." In case this increase is made, the extra surgeon could be selected for his surgical ability.

"Of the nearly nine thousand troops at one time or another encamped at Framingham during the summer, only

one serious case of illness occurred. This seems a remarkable record, in view of the great amount of sickness of the U. S. Volunteer soldiers in camps Alger, Va., Thomas, Tenn., and in camps in Florida. This, I believe, can only be accounted for by the supposition that the sanitary conditions were better at Framingham than at the camps mentioned, and that there was a better supply of pure water; this was probably true." (S. G. Report, 1898.)

Emergency lectures following the course adopted by the Mass. Emergency and Hygiene Ass'n, have been given with more or less regularity to the different organizations of the militia the past seven or eight years. The Association has furnished skeleton charts, etc., in fact everything required for a course of instruction in first aid to the sick and injured; it has also furnished diplomas to soldiers who have passed satisfactory examinations and have been recommended by their medical officers. Too much praise cannot be given this noble society for its good work. Prevention of much suffering, and even loss of life amongst Mass. militiamen, were due to the instruction received in first aid by the militia of the Commonwealth. Dr. Otis H. Marion has done much to advance this work in the militia.

To this course I would add instruction in camp hygiene, the prevention of contagious disease, etc., embracing policing of camp; personal cleanliness; the importance of reporting venereal disease to prevent its spread by drinking vessels and other means; care of water supply to prevent pollution; bathing, clothing, ivy poisoning, and kindred subjects.

No rules and regulations to prevent disease can be carried out without the intelligent co-operation of officers and enlisted men. An officer high in command in U. S. V. told me that he saw a soldier lying on the ground and drinking from a stream that had been contaminated nearer its source by washing, bathing, etc. There was no need of this

individual endangering his health by drinking from the polluted stream; there was an abundant supply of pure water in each regiment. Such reckless individuals form a small part of the volunteer force; the great majority are earnest, patriotic men who are anxious and willing to learn and practise everything which conduces to their physical welfare. For years I have believed in the importance of individual instruction to officers and men. With this end in view I advocated and, with the permission of the Surgeon of the Ninth Mass., gave instructions to officers and men in the above-mentioned subjects. Each day the members of one company were instructed, until the twelve companies of the regiment had been educated on these important matters. The fact that disease was more to be feared than bullets, was impressed on their minds and with good results. In two months' service in this country, there was not a single case of typhoid in this regiment, in fact I don't think it can be justly said that Mass. brought any of the typhoid cases to the south.

This Ninth Regiment, according to Lieut. Col. Weaver, the regular army officer who mustered it into the U. S. service, was an exceptionally fine body of men physically. Every care was taken to prevent disease, but while in Cuba the hardships during the siege of Santiago were so great that it was impossible to carry out measures for the prevention of disease.

Since writing this paper I have seen quotations in newspapers and medical journals, from high medical authorities, advocating the education of line officers in matters pertaining to camp hygiene. I have the satisfaction of knowing that I have not only advocated, but have practised it in the late war and with results that have demonstrated its importance.

The ration must be improved upon and adapted to the tropics. Expansion means an army in tropical regions, and conse-

quently it will have to adapt itself to the surroundings. This hardly comes within the scope of this paper. No doubt the military experience of our troops in the Phillipines, Porto Rico and Cuba has taught this lesson, and in the near future we shall probably see a ration adopted that will fully meet the demands. The U. S. Army ration as it stands to-day has stood the test of experience for many years in this country, and I believe gave good satisfaction in camps of instruction.

By direction of President McKinley a special order regarding the food supply of the United States army in the tropics was recently sent to Commanding Generals at Manila, Havana, Cuba and San Juan, in order to determine what alterations, if any, in the established rations would be conducive to the better health of troops serving in tropical countries. Each of these officers is directed to institute a board of three officers serving respectively in the line, medical, and subsistence departments, to examine into the subject of any desired changes in the ration for the troops serving in his command, and to cable the results of such inquiry to the Secretary of War. It is fair to presume that this will result in great improvement in rations for our troops in tropical climates.

For many years this State has maintained a well organized ambulance corps under the command of an able and efficient officer.

The commencement of hostilities found this government with no provision made to take this or other like organizations into its service.

To prevent the possibility of again losing the valuable services of such well trained men, I would suggest that one or two men be detailed from each company of each regiment for instruction in ambulance corps. I would also deem it advisable to consider the value of a corps of trained female nurses in connection with the militia.

Perhaps there is no position in the army more unsatisfactory than the surgeon's; he has no power to command; everything must be done with the approval of his commanding officer; he is censured for the health of the command, when perhaps he has made every effort to do his duty. Certainly this State has a medical service of which it can feel proud. The examinations for admission to the medical department of the Mass. Vol. Militia since 1874 have been very strict, embracing the following subjects: anatomy, physiology, chemistry, materia medica, theory and practice, surgery and hygiene. To this has been added a physical examination. Our staff is composed of men who have been tried and found successful in their chosen profession; they have given to the work the requisite time and study which it demanded. And will you believe that their training has been for naught? That those who have studied and prepared themselves for this emergency have failed? To believe this is to be unreasonable and disloyal to our profession. The volunteer surgeon who was experienced in military medicine before the war, who was earnest in his calling, could not fail to do good work when summoned to active service.

Considering the fact that the present generation of medical officers has seen no active service, that the large volunteer army was raised at such short notice, it is not surprising that there was something lacking it its management.

The total number of deaths reported in our army, including regulars and volunteers, from May 1st, 1898, to April 30th, 1899, is 6,406. Of these 5,438 died of disease, and 968 were killed in battle or died of wounds, injuries or accidents. This statement makes a great impression as to the danger of disease, rather than of bullets; but it is only fair to consider that an army of nearly a quarter of a million of men was exposed to disease for twelve months

where only a small portion of this army was exposed to bullets for a few months. However, considering this, it shows that the prevention of disease is a very important consideration in dealing with the health and effectiveness of an army. Had the entire army been engaged in active warfare, of course the percentage of deaths from wounds would have been much greater.

Petty bickerings between regular and volunteer surgeons, trying to shift responsibility from one to the other, only tends to lower both in the eyes of their fellow practitioners and the laity. Let them come together even more closely than formerly. Let them work together for the public good and remember above all things that they are members of the same profession. To say that regular army surgeons are not bright and intelligent physicians as a class, is unreasonable. The examination that they pass for admission to the Medical Corps is sufficient to guarantee this; to say that volunteers are ignorant and inexperienced is to belittle the whole profession and degrade it in the eyes of the people. They both are lacking in some qualifications to make the best type of medical officers; many of the volunteers are lacking in military medicine, particularly as regards red tape, etc., and the regular in experience in general medicine and surgery. The combination of the regular and volunteer would make the ideal medical officer. If the regular profits by his experience in this war, and the volunteer by his, and they work together harmoniously in the future, the question of good army medical service will be solved. Let us not give way to personal feelings, criticising physicians who have done the best they could under the cirumstances, and who have perhaps done better than we could under the same conditions. Let us note our imperfections and try to improve the service for those who succeed us. Contributions furnished by experience will be a great aid to our successors. Exaggerated and sensational accounts of mismanagement do no good.

Some of the important improvements in our State service the past five years are as follows:

I. Physical examination of recruits; at first voluntary and now made mandatory.

II. Ration to be used at camp. Something after the pattern of army ration but modified for adaptation to State service.

III. School for Medical Officers. One session annnally.

IV. Company Bearers; four privates in each company specially trained in emergency and first aid work.

V. System of red tape patterned after regular army, but modified for State service.

I have merely touched upon certain suggestions which could well bear elaboration, but time and space forbid.

The following is a brief résumé of the suggestions offered to improve the efficiency of the army medical service. Some of them have been practically adopted by the Mass. Vol. Militia, and it is hoped will be adopted by the entire National Guard.

I. That Professorships of Military Medicine be established in every reputable Medical College.

II. That until such time as Professorships are established, the State provide a course on required subjects.

III. That physical examination of officers and men be made mandatory.

IV. That troops at State encampments conform as nearly as possible to duties required in service on the field.

V. That every medical officer receive commission. (This pertains to contract surgeons.)

VI. That one or more men in every company be detailed for regular instructions in ambulance corps, to prevent possibility in the future of the country losing the service of men especially trained for this service.

VII. That one Medical Officer be selected in each regiment or brigade for his surgical ability.

VIII. That a corps of trained female nurses be organized in every State.

IX. That Divisional Hospitals, in the broad sense, be abolished, and that small Brigade and Division Hospitals be established for special cases, such as surgical and contagious.

X. That the volunteers follow, as closely as practicable, the regular army, so that when called into service together the two branches may work in harmony.

XI. In addition to the present instruction in "First Aid," a course in prevention of contagious diseases, etc., etc., be given.

XII. That a reserve staff of medical officers be formed in each State; said staff to be composed of ex-members of the medical department of the National Guard.

ARTICLE IV.

# VOLUNTEER AID WORK IN FOREIGN TERRITORY.

By ELLIOTT G. BRACKETT, M.D.
OF BOSTON.

READ JUNE 14, 1899.

# VOLUNTEER AID WORK IN FOREIGN TERRITORY.

In responding to the request of your committee to make a few remarks on the volunteer aid work of last summer, it seemed best to restrict such to some of the general considerations, which would suggest themselves, from an opportunity of seeing this work conducted under unusual conditions. From this standpoint the position of bodies of volunteer relief, particularly in these days of well equipped facilities for meeting emergencies, is a question of great interest.

The work of the Massachusetts Volunteer Aid Association of last summer is so well known to you all—probably the majority of those present were more or less intimately connected with its affairs—that I do not intend to take your time with the details of any special department. It came into existence with the urgent demand at the time, filled its mission, and then was over. But now that it is a thing of the past, and yet is not too far distant for us to lose the active interest in its affairs, and what it accomplished, there is much we can learn from the retrospect of the experiences during this time, and it is my object to briefly consider from this point of view, the results of my connection with this Association in its work in Santiago.

Toward the latter part of July, the officers of the Volunteer Aid saw the need of having in Cuba a representative of the Association, who should have charge of the distribution of the supplies, determine from personal observation what

needs of the men at the front could be reached by this society, and act as the representative in its work out of the country. Many supplies which were fast collecting here had already been sent, but they were not in charge of any one, either on the way or after their arrival, and it was doubtful if they would reach their intended destination or receive the proper distribution; and these doubts proved to be well founded, for in the condition of affairs at that period every one had more than enough of his own work to do, and could not be expected to give attention to other affairs, requiring so much time and attention. It was this position it became my privilege to take.

The plan was to send such supplies as were at hand to Santiago, to have them distributed directly to the regiments, according to the special needs, and to personally determine what might be the special requirements of the men at the time, which could be met by an independent organization. The real state of affairs at this time was, as you all know, much a matter of conjecture. Information was meagre and unreliable, but that there were distress and suffering, and particularly among the men who were not hardened by long training, was certain. It was known that sickness had appeared, and was on the increase, that it was approaching the rainy season, the worst of the year, in a province which had always been infected with contagious disease, and these conditions, combined with the fierce heat of the tropics, and the presence of so many troops crowded together, left no room for doubt of the need of aid in a general way. With these as facts, were rumors of scarcity of so many necessary supplies, and although the probable exaggeration was recognized by all, the indication of the need of aid was plain. Until further information could be obtained, it was proposed to send such supplies as would be needed by the sick and convalescent, in the camps and in hospitals, both in the way of foods and clothing, while the

special needs could be determined after arrival, and procured if such were possible.

The problem of reaching Santiago, and in the shortest time possible, was met by the officers of the Association in a fortunate way. At this time the province was closed to ordinary travel, no steamers except a few of the government transports were in this part of Cuba, and quarantine had been declared against the port; therefore, it became necessary to reach the destination by an indirect route. Through the courtesy of the Boston Fruit Company, the supplies were sent, and I took passage to Port Antonio, Jamaica, 100 miles south of this part of Cuba, and from thence a small sailing vessel was chartered to complete the journey to Santiago. In this way the errand reached its destination early in August, a little over ten days from the time it was first considered as a plan.

Here, the conditions under which the men had been placed during the previous two months gives one the best idea of the position of an organization, such as the Volunteer Aid, in its effort to render assistance so far from home, and this applied particularly to the men of the volunteer troops, for whom the work was originally, but not solely intended, who had not the opportunity to learn from experience, or from training, how to save their energy, and avoid many dangers under conditions so unusual and trying, even to veterans, and who would in consequence suffer more from the results of their exposures.

Without time for acclimation, these men had landed, after nearly two weeks' confinement on a crowded transport, had marched through an unusually rough and hard country, were two days in the fight, and for three weeks immediately following, lived in the trenches for most of the time with no shelter either from the sun or the almost deadly rains. During this period they were on the short rations which their difficult position necessitated, and exposed during the day

to the intense heat of a tropical midsummer, and at night to the damp chill found in these latitudes. From the time of the surrender of the city, they remained camped in the small shelter tents under practically the same conditions. By the early part of August the prolonged exposures, heat, and unusual living had begun to show itself extensively in the very great debility among the men in general, and the breaking out of diseases. Fortunately the most prevalent were the forms of the climatic disorders, rather than the contagious diseases, but the effect of this severe type of malaria on the men already much weakened by the conditions of the previous two months, was much more disastrous than it would have been under any other conditions. There was no provision for the convalescents, either as regards place or food, and since the fever usually produced persistent digestive disorders, the recovery was extremely slow, and subject to relapses, and this long absence from the ranks threw additional burdens on the remaining well. Added to this physical debility was the uncertainty of their detention on the Island, and this with the knowledge that their work as troops was practically over, that they were merely waiting their turn to be taken home, and with the increasing disease, which daily thinned the ranks of the men in active service, made the condition seem still more anxious, when the question of prolonging the stay was considered, and this in a city where the usual epidemic of yellow fever was thought by all to have already begun, and with conditions ripe for its development. It was in such a situation, the result of the sudden culmination of events, that properly directed aid from an independent organization could be of great material help to tide over the emergency, although whatever one branch might accomplish could be but a comparative mite in the presence of such general need. The emergency which the officers in that city had to meet was an unusual one, both in character and proportion. The

amount to be done was enormous, the conditions both of place and season difficult to combat, the facilities meagre, and the corps of workers small and already weakened by their hardships. It can be truly said that the improvement which took place, and the low mortality which finally resulted, was due alone to the strenuous efforts of the officials left in charge, in spite of their handicapped position, and of the lack of early aid in the way of suitable supplies and means from headquarters in this country.

In such an emergency the most important needs must receive first consideration, for something must give preference. So far as the troops are concerned, the sick must be attended to as best facilities will allow, the body of troops which make the working force must be cared for, and the convalescent sick must receive later attention. This is not neglect, but the only solution of the difficulty, and in times like this the independent organizations find their work waiting for them. It is to fill in the gaps, which in the sudden emergency cannot be or are not provided for, and from the position far away from home cannot be filled without disastrous delay. The work naturally has but little to do with the well; such is not necessary, and would interfere with the system so necessary to maintain proper discipline.

I shall not enter into the detail of the work of the relief of this department during last summer. It would be enumeration of days of tiresome routine. The character of the work to be done and the direction in which to put it forth was only too evident. It remained only to do what was possible with the limited material and the conflicting circumstances of place and facilities, trusting that the effort would meet with success. Fortunately the stay of the volunteer troops in this section was not prolonged.

The relief work in a territory so remote from any base of supplies, must consist in using material at hand to the best

advantage; the supply will be short, and economy is necessary. The difficult problem is that of the means of transportation, and the method of distribution, for one ought not depend upon aid from those whom the object of the errand is to help, and who are already overburdened with their own duties of daily routine. The situation shows definitely along what lines the working of an organization of this kind should be carried on, without conflicting in any way with the regular service, but rendering its aid where it is needed and welcomed.

The position of relief bodies has occasionally had criticism from various sources, too often I fear by those who in the time of stress have received their bounty. The criticism without a doubt confuses the question of the need of the existence of such bodies, with the time that their work is needed, for so long as such campaigns as that of the past year are instituted, and so long as volunteer troops are used, there will be a time in campaigns when the work of such bodies will be welcomed to prevent avoidable suffering, and such loss of life as is not an incident to the active part of a campaign, for during campaigns of recent years, the usefulness of such work has been shown too clearly, and although now with the advance in the departments of science, which shorten time and space, allowing emergencies to be met with greater ease and promptness, its work becomes more defined, but none the less important.

The general operation of an organization of volunteer work in a campaign in foreign territory must be considered from a different point of view, from the distribution of relief at home within easy access of supervision, and of supplies of all kinds. An expedition of invasion would naturally have its own provision for the prevention and relief of these same conditions, and with time for preparation in these days, such would undoubtedly be done, and we may consider that there is a time when there will be the

demand for volunteer aid, and such is in the early part of a campaign, particularly if begun as most are, with insufficient time for complete preparation, when the attention of the service is directed to providing for the certain requirements, and the possibilities which pertain to comforts of sick and requirements of convalescents, and of affairs which do not affect the regular service, which cannot receive the same attention, especially when arising from a condition of so complete unexpectedness.

Therefore, the work of such organizations is needed to supply the emergency needs, which take time to collect, and individual attention to properly distribute, and particularly during this early period, when there are conditions to be met which would not be found at a later time, and such was so clearly illustrated in the latter part of last summer. To new men, unused to the hardships, and in the waiting part of a campaign, and of whom it is not asked to become regulars or veterans, the advisability of mitigating some of the rigors of campaign life is evident, rather than sacrifice men to the hardening process, for the sake of the training alone. The situation calls for requirements not provided for in the haste of departure, or perhaps not belonging to the regular service, and in no way is this shown so distinctly as in the case of convalescents. There are some kinds of work, notably among such are those of humanitarian nature, which require individualism, and when carried on by too large organizations, lose this essential by too much of detailed duties. Volunteer work also has the advantage of being unrestricted by the slow-acting and cumbersome regulations, which are the necessity of a large organization, and is able in consequence to insure that relief may reach its destination promptly, and without the otherwise certain delay. It is necessary that a relief expedition should be complete in itself, that an independent source of relief supply should be maintained, with such provision for its distribution that

demands on it can be met without delay. The particular need for this period and for this kind of work is for not only what may be classed as comforts of the sick, but for the more general care of the convalescents.

The method of procedure must vary with the different instances, but the safe rule is to reach the men through official lines, and in the direction indicated by them. In positions like those of last summer, the maintenance of army organization and system is unquestionably essential; the greater the emergency and the difficulties, the more important this becomes. By working in this way all possible conflict with personalities or with regulations are avoided, and the danger of preference is not to be feared. Aid offered in this way was always willingly accepted and appreciated, and gave the assurance of reaching where it was most needed, rather than to the most apparent distress, which too often would be the nearest at hand. An official in charge, knowing the conditions and the needs of the whole, has the best opportunity of pointing out the place of greatest demand. A commissary who is daily searching for the required stores for the camp knows better than any one the kind of foods most needed and where; and the surgeon knows the requirements of his hospital outfit, whether medicine or foods or special hospital supplies. In this distribution of supplies, when from the distance from home, and the hurry of departure, economy is most essential, this care in distribution becomes most important to a successful undertaking.

Viewed from the experience of last summer, in order that this work should be carried on successfully, it is necessary that:—

*a*. It should be directed toward supplying such needs as have resulted from the confusion and hurry in the initiating of a large campaign.

*b*. It must be carried on with deference to the judgment

of the regular officers in the command, and should be particularly directed to supply the needs which have been found lacking, and which they better than any others are in a position to appreciate.

*c*. The work must be accomplished without call on the regular service for aid, either in transportation or distribution; that is, it must be a stream of supply from an independent source, and one whose presence is felt only by the relief it brings.

ARTICLE V.

# A HOSPITAL CLEARING HOUSE.

By CLARENCE J. BLAKE, M.D.
OF BOSTON.

READ JUNE 14, 1899.

# A HOSPITAL CLEARING HOUSE.

With the aggregation of population in cities, the multiplication of facilities for suburban transportation and the growth of urban hospitals, there comes more and more urgently to the administrators of trusts for medical relief the question of making such provisional classification of applicants, in out-patient departments especially, as shall assure to the worthy poor, for whom these institutions are principally maintained, the full mede of time and attention which is their due.

From a business standpoint the hospital may be defined as an institution in which capital and skilled labor combine to provide such members of the community as are temporarily disabled and without means of support, with the maintenance and care which shall fit them to become again self-supporting and active community factors.

The hospital is a free repair shop for human machines and the capitalist who contributes to its support does so with the basal, though perhaps not with the defined understanding, that his contribution is returned to him through the community, in the lesser number of incapacitated and dependent machines; while the doctor, who furnishes the skilled labor contribution, finds his return not only in the same manner as the capitalist citizen but also in many other ways which need not be enumerated before this Society and which enure greatly to his own well-being and usefulness.

It is incumbent upon both these contributors to see that their contributions are justly disposed.

The ability to contribute, in itself constitutes a trust.

It will be seen that the foregoing statement leaves out of consideration many of the motives which have led to the foundation of, and continuance of contributions to the support of, our hospitals; not that these motives, or the sentiment underlying them, which is the vital stimulus of all human brotherhood, is decried, but that the care of, and provision for, the needy sick is so serious a business as to demand the best judgment we are able to bring to bear upon it, and often the fittest use that we can make of a cherished sentiment is to split it into kindling wood and with it start the fire in the harder fuel of our best endeavor.

Setting aside the emergency cases, whose urgent need of help is paramount to every other consideration, the applicants for hospital relief whose pecuniary fitness has to be considered, are broadly divisible into two classes: 1, those taken into the wards for more or less continued treatment as inmates; 2, ambulant cases, patients who, residing in their own homes, come to the hospital for occasional or for daily treatment. The latter make by far the greater number, and it is in response to their needs that the so-called out-patient departments of our hospitals have grown to their present dimensions and have come to require administrative effort in classification far beyond anything that would be needed in a hospital receiving house cases alone, for with a patient admitted to the house there is usually ample opportunity for leisurely investigation.

That the sifting out process in the admitting room of the out-patient department of a large hospital, however, is one which must be done with precision and celerity is shown not only by the large number of ambulant patients applying for relief, but by the number of sub-divisions which must be made in order to secure to the eligible patient in the waiting room protection, not only from the presence of contagious diseases but from the encroachments of those pecuniarily

unfit applicants, who, either through ignorance or guile, rob, not so much the institution and the doctor as the really needy.

The "pecuniarily unfit" cases in out-patient departments in large cities may be generally estimated to constitute from five to ten per cent. of all patients applying for treatment and they may usually be classified as follows: 1, persons ignorant of hospital usage and both able and willing to pay for professional services rendered; 2, persons who by a pretence of poverty seek to procure medical services for which they are able to pay; 3, the class of persons known in institutions' departments as "rounders," people who go from one hospital to another seeking advice and treatment, a species of medical medicants; 4, persons who are able to pay a small fee, but who are sent to the hospital by their family physician instead of being referred to an individual practitioner.

That the people who come under these four classes should be allowed to share indiscriminately with the proper objects of charity the hospital bounty, is a lapse of justice in two directions, justice to those who support the hospital for a definite charitable purpose and justice to the doctor and the patient, for, with the large number of truly worthy patients, the time which the doctor can afford to give to the hospital is none too much to be divided among the many, and with the increasing difficulty of gaining a remunerative foothold in private practice the doctor, and especially the younger doctor, should have the fee, however small it may be, which the patient is able to pay for service rendered.

Various suggestions have been made as to the best method of dealing with the "pecuniarily unfit"; one, that an already existing institution like the Associated Charities should be asked to establish a department, or to use some of its already operative machinery for the investigation and detection of these cases; another, that the doctor who treats

the patient in the hospital out-patient clinic should himself question the patient as to his pecuniary condition.

To this latter proposition there are two very evident objections, namely: that the process of questioning takes time which properly, and under the rules of the hospital, belongs to the worthy patient, and also that there should be no connection whatever between the private practice of the physician for which he is paid, and the practice in which he gives his services under the auspices of the hospital of which he is one of the recognized staff.

Against the first proposition it may be urged that while the Associated Charities organization and its records may be occasionally availed of, the duty of protecting their dependents and patients belongs properly to the hospitals themselves, either individually or both individually and through the medium of some organization in which they they shall all have part and to the support of which they shall all contribute.

The extent to which individual supervision is exercised in this city and the opinions in this matter of those having hospital administration in charge are perhaps best set forth in the following communications.

The first is a letter from Dr. Farrar Cobb, Superintendent of the Massachusetts Charitable Eye and Ear Infirmary:

Dear Dr. Blake:—You asked me to write you in regard to the system in use at the Infirmary with respect to weeding out unfit cases. Allow me to say in the first place that it is very exceptional for any but poor and needy patients to obtain admittance into the wards of the Infirmary. The questioning and investigation of all the cases recommended for admission to the hospital is especially thorough, and after careful observation of the inmates of our wards for a long space of time, I am convinced that it is rare to find a patient who is not deserving of the charity of the Institution. There have been, however, occasional instances of deliberate misrepresentation, and consequent admission to the hospital, but such cases are very rare.

In regard to the attendance in the Out-patient Department; we have for three years carried out the following method. The registrars in the Out-patient Department are instructed to question any case which they think from appearance, or address, or occupation is able to pay a physician two or three dollars, and call attention to the fact that the Institution is only for the needy poor. In some instances each day it is necessary to send troublesome cases to the Executive Offices, and have them more carefully questioned. The limit of our questioning is to ask a suspected case whether he is willing to sign an affidavit that he is an object of charity. If the answer is yes, no further questioning is done. The registration slips of each case that has been questioned at the office are stamped with one of the two rubber stamps which I enclose,* and put on file in the records. The system as it is now working is not very successful. I have no doubt that a considerable number of unfit cases receive treatment each day in our Out-patient Clinics. It is my wish within a week or two to put one of my best and most responsible clerks in charge of a *special inquiry* room in the Out-patient Department for two hours each day, and to have all cases suspected to be able to pay sent to him for thorough questioning.

One thing that I especially object to on theoretical grounds is the questioning of the patients by the doctors themselves, especially in the presence of other patients. I am decidedly of the opinion that such practice is wrong, and liable to misinterpretation. Investigation and questioning should be done by the Executive Department through some responsible agent. As I said above, now that our new Infirmary is running successfully I hope within a week or two to make a radical departure in the line suggested.

Yours very truly,

FARRAR COBB.

At the Boston City Hospital, out-patients are refused treatment by the physician in charge of the department at which the patient applies, if the physician thinks the case in question unsuitable for charity. If the patient insists upon being treated after being refused treatment upon the grounds

* "This case was unfit and therefore was refused treatment."
"This case has been questioned at the office and is a suitable case."

of pecuniary unfitness the question may be referred to the Superintendent for further investigation and decision. In the eye department there is a rule that the patient asking for a prescription for gold rimmed glasses shall be refused treatment on the ground that the ability to afford this luxury implies ability to pay a physician. Dr. Rowe may be quoted as saying: "I do not believe that abuse can be stopped until all hospitals in all cities agree on some simple rational system and live up to it."

At the Massachusetts General Hospital all new out-patients are seen by the admitting physician and questioned as to their ability to pay for medical treatment, unless their appearance evidently indicates that they are suitable cases for charity; patients who admit their ability to pay something are refused treatment; those in regard to whom the admitting physician is in doubt are referred to the Superintendent for further questioning and final decision.

Patients known to have been treated in other hospitals for the trouble for which they come to the Massachusetts General Hospital are referred back to the original clinic for treatment unless there seems to be a good reason for retaining them.

At the Boston Dispensary all new patients, except such as are evidently charity cases, are questioned as to their ability to pay for treatment. Doubtful cases which it seems best to admit are reported to the physician who is to have charge of the case, for supplementary questioning.

In one way or another therefore the hospital administrations seek to winnow their applicants at the gate, but the winnowing process is one which lacks uniformity and co-operation and hence fails of the best execution.

The use of the term Clearing House in this connection is not inadvisedly made, for in addition to its limited application in the banking system as early as the year 1856 in this country, a clearing house in other lines of business

has come to be a central bureau not only for the establishment and maintenance of balances but for such co-operative investigation as may constitute it a bureau of information as well; and it is readily conceivable that the establishment and maintenance by hospitals in a large city of an officer to whom, or a bureau to which, there could be referred, for more leisurely investigation, pecuniarily doubtful cases, would not only relieve the individual administrator of a part of a most unwelcome task, but would in time become a channel through which there should flow interchange of ideas, and from which there should emanate to the public instructive information as to the rights as well as the obligations of hospitals.

Such an officer or bureau constituting a clearing house should be under the direction of a Board of Control, said Board of Control being, not as is proposed in the new Dispensary Bill of New York, the State Board of Charities, or other alien organization to which is given a dominant power, but a Board of Control made up primarily of the medical superintendents of the hospitals themselves. A Board thus formed and backed if possible by Legislative enactment, making fraudulent abuse of medical charity a misdemeanor, would be likely to be an efficient piece of machinery for the attainment of the desired end.

The attention given to this question of the abuse of medical charities in the meetings of the National Association of Charities and Correction in recent years and the agitation accompanying the framing of the Dispensary Bill in New York, are among many evidences that this subject is attracting the thoughtful consideration of the public at large, and the conclusion that any carefully considered remedial measure emanating from medical sources is likely to receive cordial support, is based upon the fact that while the abuse is one which concerns the well-being of the community as a whole, its correction is properly one of the responsibilities of the medical profession.

ARTICLE VI.

# THE VALUE OF THE PULSE IN DIAGNOSIS AND PROGNOSIS.

By HENRY JACKSON, M.D.
OF BOSTON.

READ JUNE 13, 1899.

# THE VALUE OF THE PULSE IN DIAGNOSIS AND PROGNOSIS.

THOUGH I have no new theory to advance as to the value of the pulse in diagnosis, yet I desire to bring the subject up at this meeting because to me it seems a matter of the greatest importance, and one perhaps that we do not consider as carefully as it deserves. The pulse is the final index of the vital capacity of the individual, and therein allows us to judge of the resistance to disease of the individual patient at a stated time: our patient may have a diseased heart, one much weakened by organic valvular disease or by degeneration of the muscular substance dependent upon disease in the heart itself or upon some toxæmia, but if the heart is able to drive the blood through the system for the time at least death is not imminent. Therefore the pulse is, as stated, the final index of what the heart can do; so long as the pulse is of fair quality the system is supplied with blood and life is possible.

In diagnosis it is not my wish to discuss the value of the pulse in the various forms of organic heart disease, as for instance the Corrigan pulse in aortic disease, and the small thready pulse of mitral stenosis, but rather to consider its value in the diagnosis of some of the more important and often obscure general diseases, especially typhoid fever, miliary tuberculosis, cerebro-spinal meningitis and other diseases of the brain and its membranes.

The pulse of typhoid fever is in uncomplicated cases not very rapid, rising somewhat with each access of fever, but

averaging 90–100 or less in cases of moderate severity which run their course without untoward accident; under prognosis I shall speak at greater length of the inestimable value of the pulse as an index of the advent of dreaded complications and the exhaustion of the patient by the toxic effects of the specific organism of the disease.

In acute tuberculosis we have a very different picture: the pulse is rapid, averaging 120 or more long before the patient reaches a critical stage of the disease. In typhoid fever the pulse may temporarily be 120, but it never remains for several days at this point unless the patient is severely sick, whereas in general tuberculosis we find great rapidity of the pulse for days, and even weeks, in a patient presenting no signs of immediate dissolution, often very comfortable and anxious to be up and about the room. The rapidity of the pulse in general tuberculosis is of much value, as in both diseases the course of the temperature may be similar; in both diseases there is an absence of leucocytosis, and often the general condition of the patient may leave us much in doubt, though in the one hebetude is usually much more marked than in the other. Of course, the Widal reaction in the one case, and tubercle bacilli in the other, may if present give us an absolute diagnosis.

In cerebo-spinal and tubercular meningitis the pulse may be almost pathognomonic; in the early stages, during the time that the vagus is stimulated by a moderate degree of irritation, the pulse is slow, or rather, what is the important point in diagnosis, is slow in proportion to the general condition of the patient. To take a case we have before us, a patient who for a day or so has had a moderate fever, who is evidently very sick, with perhaps vomiting and severe headaches; and yet we find a pulse of 70–90. If the case were typhoid fever the pulse would be more rapid; the slow pulse in the case under consideration is, however, slow because of the direct action of the products of inflammation

on the vagus. Therefore, the pulse is no criterion of the general condition of the patient. If there is added to the slow rate an occasional intermittency, we have a still greater assurance that the pulse is dependent upon some direct pathological lesion, and can by no means be considered as an index that our patient is not seriously ill as is suggested by his general condition.

It is not an invariable rule that the pulse is slow in the early stages of meningeal trouble, but if not continuously slow we find that the rate varies much without any corresponding change in the condition of the patient and independently of the degree of the fever. I would cite two cases seen in consultation, illustrative of the value of the pulse in meningitis. Several years ago I saw in consultation a young child who had been sick several days, was somewhat stupid, had vomited, had fever, and seemed to be very sick: the pulse was one hundred, and intermitted occasionally. I suggested tubercular meningitis as the most probable diagnosis, chiefly perhaps on account of the pulse, a symptom that the attending physician had looked upon rather as his main stay in hoping for a favorable issue of the case. My diagnosis was correct. On the other hand I saw this winter a case where cerebro-spinal was certainly a possible diagnosis, and yet a rapid, regular pulse made me very doubtful as to the correctness of the diagnosis, and complete recovery in a few days convinced me of the truth of my impressions.

The slow pulse is only seen in the early stages of meningitis, as later in the course of the disease, when the vagus has been paralyzed by pressure or by disease of the nerve itself, we find an extremely rapid pulse as the inhibitory action of the nerve has been destroyed; to use a metaphor, the engine runs with the throttle wide open.

In cerebral hemorrhage the pulse may again be of great value, when definite symptoms are wanting, as paralysis and

allied phenomena; that is to say, in cases brought to a hospital in a comatose or semi-comatose condition, cases so aptly described by Mr. Ernest Hart as "drunk or dying." In alcoholism the pulse is rapid, in cerebral hemorrhage slow; and hereby, as in a case I wish to cite, we may make our diagnosis. During my last service a man was brought into my ward by the police: he was unconscious, was tossing about the bed, was flushed. The only history obtainable was that two days previously he was found in a doorway, taken to the station house where the diagnosis of intoxication was made, then was removed to the city prison, whence he came to the hospital. Careful physical examination showed no fracture of the skull, no evidence of external trauma; strong stimulation of the orbital nerve at its exit gave evidence of a slight left facial paralysis and the left side was moved less vigorously than the right side; the neck was a little stiff. Leucocytes, 12,800. Lumbar puncture negative: temperature 99°. His pulse was forty, a factor that to my mind rendered simple alcoholism impossible. The evidence of paralysis was slight and not easy to obtain, the pulse was evident. The man died the day he entered, and autopsy showed fracture of the skull and extensive laceration of the brain, with subdural hæmorrhage. In view of the diagnosis that I made, I was criticized because I had not suggested trephining; I considered the matter and decided against it because the severe general symptoms without marked paralysis made it evident to me that the man was suffering from extensive cerebral trouble, rather than from total destruction of a limited area which might be amenable to operative procedure.

Dr. Bowditch has recently called attention to the value of a rapid pulse in the diagnosis of early tubercular trouble. On the other hand an extremely rapid pulse without evidence of serious disease may be suggestive of exophthalmic goitre as in a case described to me in which later goitre developed.

In many other diseases the pulse may assist us in forming our diagnosis, as for instance in the early diagnosis of acute follicular tonsillitis and diphtheria, the one usually associated with high fever and rapid pulse, the other in the beginning often giving rise to only slight fever, while the pulse may not be markedly quickened unless toximia has already taken a severe hold upon the patient.

I have spoken of the value of the pulse in the diagnosis of typhoid fever, but in prognosis we have in the pulse what I believe to be an infallible guide. In uncomplicated cases the rate of the pulse is slow relatively to the degree of the fever and the general condition of the patient. When the pulse is continuously rapid, the prognosis becomes proportionately grave with the increase in the rate, though no other alarming symptoms arise to render us doubtful as to the outcome of the case. Leaving out of consideration hæmorrhage and perforation, a rapid pulse is an index that the patient is in danger of succumbing to the specific toximia produced by the disease. The pulse is to us an index of the effect upon the individual patient of the poison of the typhoid, and is of far greater value than any other one symptom or any group of signs or symptoms.

In my early student days I read an article, I think by Dr. Wilson of Philadelphia, stating that in typhoid fever the danger line was reached when the pulse registered 120; and all cases that I have seen since that day have only confirmed in my mind the great dependence to be placed upon this axiom. I have seen patients die who had only a rapid pulse as indicative of danger, while again and again I have seen patients recover where the pulse ran from 110–120, yet they were very stupid, had involuntary passage of urine and fæces and perhaps frequent vomiting. I am so sure of my ground in this matter that in the individual case I dare to make a favorable prognosis if the pulse is slow, no matter how unfavorable the other symptoms may be. On the

other hand a rapid pulse, independent of any complication as hæmorrhage or pneumonia, causes me great anxiety.

After careful consideration of this matter in a considerable number of cases, I feel convinced that the danger line is reached when the pulse becomes 120. Of course many cases recover when the pulse is rapid, but not without a severe struggle for life.

The remarks which I make as to the prognosis in typhoid do not refer to cases complicated by intermittent diseases or by perforation and hæmorrhage. In such instances the value of the pulse lies rather in diagnosis than in prognosis.

A sudden rise of the pulse without apparent cause suggests hæmorrhage, especially if accompanied by a fall in the temperature. In this connection we must remember that the fever may fall by crisis, but in such instances with the fall of temperature comes a corresponding fall in the pulse, and the general condition of the patient is decidedly favorable.

In perforation the pulse is of the greatest value, as in the following case. Last summer, at the time of my visit, one of my patients was perfectly comfortable with a pulse of 80: at half-past one my house physician, Dr. Stevens, telephoned to me that the man had a pulse rising rapidly, had pain in the right side of the abdomen, an access of fever with an expression of anxiety. Examination showed no blood in the bowel, the pulse was 120, there was a leucocytosis of 15,000, and there was rigidity of the right side of the abdomen. The surgeon who saw him in consultation agreed with me in the diagnois and found a perforation when he operated at four o'clock. In this case the leucocytosis was of great value, as I have never seen leucocytosis in an uncomplicated case of typhoid fever. Hæmorrhage was ruled out by the rise in temperature, and the absence of blood by rectal examination.

The pulse may be rapid in typhoid fever in other complications, as otitis media and phlebitis, without rendering the prognosis unfavorable; whereas in acute inflammation of the parotid the prognosis is grave, more so I think than when pneumonia or pleurisy is the complicating pathological factor.

In convalesence the pulse is often rapid, and then of no serious import; it is the rule that a patient will pass through a severe typhoid with a pulse not more than 110, and when he first begins to get about have a pulse of 120 or much higher, without the slightest indication that we have any serious danger to combat. Further, many slight troubles in convalesence, as constipation, excess in diet, fatigue from seeing friends, cause a sharp rise in the pulse.

I have once seen a slow but very poor pulse in typhoid in a man that was seriously sick; examination of the heart showed that the pulse was not a sufficient index of the action of the heart, in that many beats were so feeble and inefficient that the wave was not transmitted to the wrist.

In the first volume of the medical and surgical reports of the Boston City Hospital is a valuable article upon typhoid fever, by Dr. Upham. He speaks of the prognostic value of the pulse and states that in sixty-five cases that recovered the average highest pulse was 108, while in fourteen fatal cases the average highest pulse was 140.

I have looked over a large number of charts of typhoid fever cases in the Boston City Hospital, and found the same relative proportion of deaths to the pulse rate.

Finally I wish to quote from Dr. C. Ellery Stedman's valuable essay on typhoid fever, published in the third series of the City Hospital reports. In this essay Dr. Stedman says, "Temperature for diagnosis is invaluable. For guidance during the disease I rely on the pulse." "I have seen no patient die whose pulse has not reached 120, twenty-four or more hours before death," and finally "In these ten

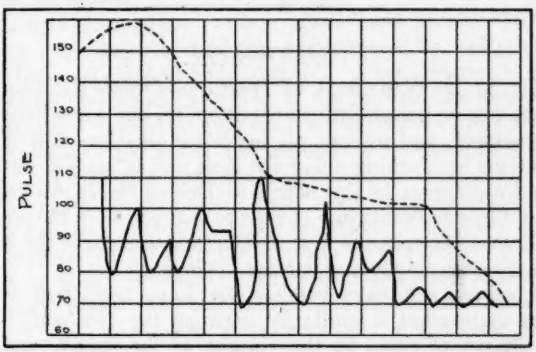
years the pulse has given the warning of approaching trouble even when the temperature has signified nothing untoward."

I wish to call attention to one very marked sign of cardiac weakness that so far as I know is not mentioned by most writers. Dr. Whittier has referred to it in a short article in the Boston Medical and Surgical Journal. Namely, a discrepancy between the rate of the heart beat and the pulse as felt at the wrist; in many cases it is difficult to count the pulse because of its irregularity or the constantly changing rhythm, though we are conscious that a feeble pulse is occasionally felt beneath the finger, but in the cases under consideration no pulse wave is felt. This condition may be said to represent the acme of cardiac weakness and may be responsible for some of the cases in which a slow pulse is reported in fatty degeneration of the heart. This discrepancy between the rate of the pulse and the apex beat I have seen frequently in great dilatation of the heart dependent upon disease of the myocardium, much less often in valvular disease of the heart. I have noticed the same phenomenon in acute degeneration of the heart as seen in infectious diseases, notably in typhoid fever and in diphtheria. In diphtheria it may be seen during the acute stage, and also in the serious cases of cardiac weakness which appear late in convalescence, perhaps dependent upon diseases of the nerves regulating the action of the heart similar to the specific neuritis which causes paralysis of the muscles of the naso pharnyx or the muscles of the extremities.

The following cases give a good illustration of the phenomenon:—

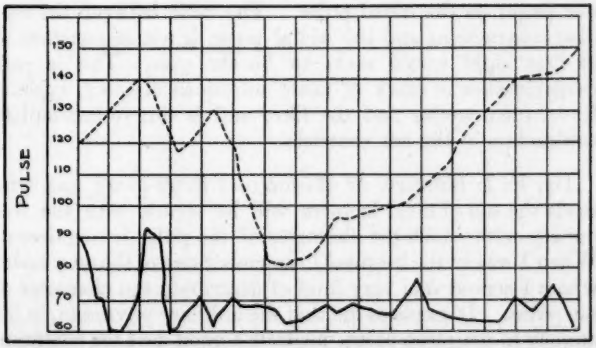
Case 1.—Man 45 years. Great alcoholic excess; short of breath for six months. Great general œdema, cyanotic. Pulse irregular, intermittent; heart dilated. At the apex was a systolic murmur. He gradually recovered and was

discharged in a month. On entrance the apex beat was 160, while the pulse was 110. For ten days the apex was much more rapid than the pulse, while later in convalescence they coincided.




Case 1.—Parietal disease of the heart. Dotted line shows the apex beat.

Case 2.—Man 45. No alcoholic excess. No rheumatism. Arteries sclerotic. Heart much dilated. Great improvement at first in hospital. Then many attacks of pulmonary infarction: acute and rapid dilatation of the heart, great œdema and death.




Case 2.—Dilatation of heart, probably parietal disease. Pulmonary infarction. Death. Dotted line shows the apex beat.

These two charts are illustrative of the condition of great weakness of the heart in which a large proportion of the beats are so feeble that no wave is transmitted to the wrist.

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## DISCUSSION.

Dr. E. N. WHITTIER, of Boston:—Doctor Jackson has made a most important contribution to our knowledge on this subject, and we can all make for ourselves still larger additions by working along the lines he has marked out. I could not expect to add much to Doctor Jackson's paper, except in one direction. I refer to diminution in the normal difference between the first and second sounds of the heart as indicating impending cardiac inefficiency.

I believe, and have believed for some time, that where there approaches a similarity between the two sounds, in the length of time, in the volume of the sound, and the quality of the sound, the heart requires stimulation to a larger degree, and compels an appeal to our utmost endeavor, more than in any other departure from normal condition of the heart, for it is of the greatest significance in cases where at the outset there were no evidences of impairment of the heart.

Also in our examinations with the stethoscope we ought more frequently, when listening to the heart sounds, to place our finger on the radial pulse. The ratio between the cardiac contractions and the radial pulse is not so constant as at first sight would seem to be the case. This is particularly seen in cases of acute infectious diseases, typhoid fever, pneumonia, and the like, and is due to incomplete contraction of the left ventricle.

Dr. F. I. KNIGHT, of Boston:—I think every one who reads the old clinical lectures will be struck with the frequency with which the character of the pulse is mentioned. When I was in the hospital I remember one of the men under whom I served was very fond of describing the character of the pulse. It seems to me that the ordinary physician, in his anxiety to diagnose cases, neglects a good deal the condition of the patient, information that we get from the study of

the character of the pulse. I remember an example where neglect of the character of the pulse resulted in a grave error. This is seen especially with regard to the abuse of the use of digitalis. Before giving this drug, the character of the pulse must first be considered. I recall one case, a lady, who came to me with a rigid, hard pulse, who was taking large doses of digitalis, administered by a physician, who believed she had heart trouble. I was obliged to give aconite to overcome the effect of digitalis on the heart. I remember a case of pneumonia, where the man had a full, rigid pulse, being given large amounts of alcoholic stimulants. This ought not to be.

In regard to diagnosis, I am very much interested in what has been said concerning the character of the pulse in typhoid fever and tuberculosis. I saw a case last week in South Boston. I believe that Doctor Whittier saw the same case. The beginning of the sickness simulated typhoid. For two weeks there was only a moderate elevation of the pulse and temperature, when suddenly the temperature rose, and the patient died within two weeks from that time of acute miliary tuberculosis. In that case the character of the pulse failed to diagnosticate the condition.

ARTICLE VII.

# PRELIMINARY NOTE ON THE PROGNOSIS OF NEPHRITIS.

By RICHARD C. CABOT, M.D.,
AND
FRANKLIN W. WHITE, M.D.
OF BOSTON.

READ JUNE 13, 1899.

# PRELIMINARY NOTE ON THE PROGNOSIS OF NEPHRITIS.

THE material upon which this study is based was obtained as follows: From the records of the Mass. General Hospital for the last twenty years, the course and symptoms of all fatal cases of nephritis were noted, and the duration of each case, from the time of the first symptom to death, was estimated as accurately as possible. In addition to this, circulars were sent to the address of every patient who had left the Hospital between 1880 and the present day suffering from nephritis of any type. By means of these circulars, we attempted to find out the condition of the patients since leaving the Hospital, and the date of death in the fatal cases. Only a portion of those to whom circulars were sent returned any answer, but the answers received were very instructive. All of the cases, diagnosed in the Hospital records as nephritis, who returned any answer to our inquiries, were followed up as carefully as possible, and in many cases the urine was examined. The number of these cases, together with the fatal ones, is 332.

In addition to these, we have a considerable body of data, sent us with great kindness, by a large number of physicians to whom we wrote, asking whether they had ever known a case of chronic nephritis to recover, and also how long they had ever known a case of nephritis to last. This data furnished us details of 91 more cases, making a total of 423 in all.

We were stimulated to make the above investigations by the indefiniteness and disagreement of the statements in the

text books on the subject of The Prognosis of Nephritis, especially in its chronic form. Many writers show a strong tendency to hedge when it comes to the question: "Does a case of chronic nephritis, properly certified as such, ever recover?" Thus, for example, Rotch in his Diseases of Children, says of the prognosis of chronic nephritis: "Some cases, having the clinical features of the disease, have apparently recovered." Delafield, in the American System of Medicine, says, under Chronic Diffuse Nephritis, "Recovery seems to be possible." Osler says that in chronic parenchymatous nephritis recovery rarely takes place in a case that has persisted for more than a year, but occasionally, in children, even when the disease has persisted for two years, symptoms disappear and recovery takes place. Senator,* speaking of chronic diffuse nephritis, says that termination in recovery is a rare exception, but undoubtedly occurs. He agrees with Rosenstein that these are usually cases, which after acute onset as a result of infectious disease or exposure, become chronic, and after one and one-half or two years, end in recovery, the urine becoming entirely normal. An entire "restitutio ad integrum" of all the kidney tissue does not occur, but this is not indispensable for complete functional recovery. He also says that death is invariably the result in interstitial nephritis where the typical contracted kidney is developed, and the heart hypertrophy is marked. Recovery (functional) is possible, and under proper treatment is not rare in the first stage, that is, as long as no sign of heart or vessel lesion has developed.

Strümpell, in his last German edition, speaking of chronic diffuse nephritis, says that complete recovery undoubtedly occurs, but is rare. By the passage of the large kidney into secondary contraction "apparent healing" may occur, in which the subjective condition of the patient is almost en-

* Senator, Erkrankungen der Nieren.

tirely satisfactory. He considers chronic interstitial nephritis incurable, though life may be prolonged without any considerable unpleasant symptoms for many years. Bartels, in Zeimssens' Encyclopædia, says that chronic nephritis is incurable after a certain time, but that cases have recovered even after months of dropsy. On the other hand, Brault, in the Traité de Medicine, says that in chronic nephritis the future is always sombre, sooner or later uræmia or a complication leads to a fatal ending. Tyson also, and Loomis, in the Twentieth Century Practice of Medicine, say that cases never recover. In Albutt's System and in Fagge, no definite statements are made on this point.

Under these circumstances, with such variety in the opinions of the foremost writers, our interest centred about the question: "Does a case of chronic nephritis, certified as such, ever recover?"

At this point, it seems best to define what we mean by chronic nephritis and by recovery.

(1) We recognize two important types of chronic nephritis: *(a)* Chronic diffuse nephritis, and *(b)* Chronic interstitial nephritis.

*(a)* By chronic diffuse nephritis, we mean a disease characterized by prolonged anasarca and effusions into the serous cavities, early and pronounced manifestation of uræmia, such as headache, vomiting and dyspnœa, vascular changes such as cardiac hypertrophy and a high tension pulse, and a pale urine usually diminished in amount, containing a large amount of albumen, with an abundance of fatty elements in the sediment.

*(b)* By chronic interstitial nephritis, we mean a disease of insidious onset and long course, with comparatively early appearance of the vascular changes above mentioned, and little or no œdema until the heart fails, with late and usually less marked uræmic symptoms, a large amount of urine, of low specific gravity, with albumen present in very small

quantities if at all, and a sediment containing usually only a few hyaline and granular casts.

(2) We consider a patient to have recovered when he has no subjective symptoms characteristic of nephritis, and passes normal urine.

Defining our terms in this way, we come now to the analysis of our cases, and taking first the data from the Massachusetts General Hospital records, we find that of the total 304 cases, in which a diagnosis of chronic nephritis stands recorded on the books or the hospital, and which we have been able to follow up till death, or till the present time, there are only 17 whose urine and general condition show that the patient has really recovered. In most of these cases, although the diagnosis of chronic nephritis stands upon the books, it is difficult to see why it should have been made. From the facts recorded, it was evident, in most of the cases, that the patient was rapidly improving, that the symptoms were of comparatively brief duration, and the diagnosis seemed unjustified. In a few cases, on the other hand, the long duration of the symptoms and the character of the urine indicate an undoubted chronic nephritis followed by a genuine recovery. The following two cases are among the more remarkable of our series.

Case 1.—Woman, age 23. Eight years ago symptoms of chronic diffuse nephritis lasting for two and one-half years, headache, vomiting, marked œdema of body, face and legs, also ascites, which was tapped twice. Urine contained ¼ to 1% albumen, and many hyaline, granular and fatty casts. For the last six years she has been perfectly well and leading an active life. The urine examined a year ago and also this year was normal, free from casts and albumen.

Case 2.—Boy, aged 16 years. Chronic diffuse nephritis followed scarlet fever at the age of eight years, lasting for four and one-half years, with headache, vomiting and ana-

sarca. The urine contained ⅛% albumin and many hyaline, granular and epithelial casts. He had several severe exacerbations. Has been perfectly well for three and one-half years, and is now at work. The urine at the present time is perfectly normal.

In answer to the question, "Have you ever known a case of chronic nephritis to recover?" 48 of the physicians who were asked said no, and 21 (about one-third of the whole number) said yes.

Among these latter answers are the following:

(1) Dr. J. M. Da Costa, of Philadelphia, writes of the case of "a young woman, eighteen years of age, who had chronic parenchymatous nephritis of about a year's duration. The disease came on gradually, though there was a statement of its having followed exposure. Dropsy was a marked symptom. There was considerable albumin in the urine, and epithelial casts. She gradually recovered entirely, got married, and became the mother of five children. She was seen last year, and her urine was found to be perfectly normal."

"In a second case, twenty years of age, seen in consultation, the disease of the kidney followed scarlet fever at the age of eight. As a boy, the patient learned to test his urine for albumin, and continued to test it until he was grown up, with the result of never finding albumin absent, though not present in any great quantity. He tested for it and found it only a few months before an exacerbation of the disease, coming on after exposure; in this the urine contained large amounts of albumin and tube casts. Under a rigid milk diet for three months, mild diuretic waters, iron, and other means, the dropsy disappeared, and his general condition greatly improved, but albumin remained. Pursuing treatment for two months more, the albumin gradually disappeared, and, though constantly tested for it (and he was no longer on a restricted diet), none was found for some

months afterward, when he was looked upon as having entirely recovered. I never heard of a return of the disease."

"In a third case, a man sixty years of age, who was dropsical for months, there was a history of the kidney disease having begun after exposure about two months before be came under observation. The urine was markedly albuminous and contained tube casts, but no blood casts or blood. Under treatment for ten weeks, the albumin was reduced to a trace, and for some little time was absent; but, a few months afterward, during a period of great family distress, it was again found in the urine, though not in large amount. Under treatment, it slowly disappeared, and, examining him three years afterward, not a trace of kidney disease could be detected. The patient subsequently died of phthisis, which was hereditary in his family."

(2) From Dr. Charles G. Stockton, of Buffalo, we received the following case:

"The case came under my observation in the autumn of 1890. The patient was a woman about 46 years old, of medium height, and weighed about 240 lbs. The exact weight could not be ascertained. She had been in poor health for several years, and for the past six months had suffered from headache, dizziness, anorexia, mental confusion, irregularity of the bowels and increasing general anasarca. The aortic second sound was relatively intense and high pitched; the apex too far to the left, not palpable owing to the fat. There were no bruits. There was an increased arterial tension.

The first recorded examination showed about 12% albumin (by bulk), with hyaline, granular and epithelial casts. A more complete examination was made a few days later when she voided, in 24 hours, 26 ounces of urine containing 27.4 gr. of urea, and albumin about 4%.

Examinations were now made daily, and it was found that the amount of albumin, by Esbach's method, was from

two to four grams per litre. The casts continued; sometimes the hyaline form predominated, at other times both finely and coarsely granular casts predominated, and occasionally large numbers of epithelial casts were seen. Under treatment on the 9th of January, 1891, only ½ gram of albumin to the litre was found, and 1,136 c.c. of urine were passed in 24 hours, and the urea had risen to 30.67 grams. This amount did not continue, and she had a number of relapses, judging from the condition of the urine. For instance, on the 14th of January, there was in 1,408 c.c. of urine two grams of albumin per litre; and the same casts. On the 25th of January, the urine had fallen to 768 c.c., 21 grams of urea, two and one-half grams of albumin per litre; no casts were found. On February 27th, albumin four grams per litre, urea 29.23 grams, a number of uric acid crystals, hyaline and epithelial casts and renal epithelium. On the 8th of March, the urea had fallen to 19 grams, albumin had risen to 4.50 grams per litre; there were no hyaline casts. On the 15th of March there were no casts found. Urinary examinations showed varying results, but on the whole about the same for some months therefrom.

In September, there were six grams of albumin to the litre, 832 c.c. of urine, 21 grams of urea; no casts found. On the 11th of November, a few casts, urea had fallen to 16 grams. From this time on the casts decreased in number and were almost altogether hyaline; albumin varied. On November 19th, there were five and one-half grams per litre; December 12th, a few casts. January 3d, 1892, urine 28 ounces, urea 22.4 grams, hyaline, slightly granular casts. July 12th, 1892, ¾ gram of albumin per litre, urea 22 grams, pus corpuscles, epithelium and a few granular casts. December 6th, 1892, more than two years after I first saw her, the albumin was .25 gram per litre, one small granular cast. May 9th, 1893, there was no albumin, urea 23.71 and no casts found. From this time

on the urea was less in amount, the urine became more abundant; albumin was found in varying quantities, usually in traces only, and occasionally hyaline and slightly granular casts. For the past three years I have found that the urine has been uniformally free from albumin and casts. The patient's general condition corresponded rather closely with the state of her urine. Her symptoms were somewhat promptly relieved by treatment, but there was more or less edema for at least a year after I first saw her. Her vision was disturbed in the beginning, but an eye examination showed that the retina was not affected. I have a report on the condition of the retina reaching back as far as 1888."

(3) From Dr. D. W. Prentiss, of Washington, the following note:

"A case of chronic parenchymatous nephritis, had been ill about six months when I first saw him. Diagnosis unmistakable. Recovered in about a year. This was 18 years ago. He is still well, and has been working at his trade ever since. Theory of recovery: that but one kidney was affected."

(4) From Dr. G. Baumgarten, of St. Louis, the following:

"In August, 1894, the patient had edema of the face and hands. In October, 1894, extensive anasarca was present; urine about normal in quantity, 1,015, 1½–2% of albumin, granular casts, leucocytes, no blood. December 25, 1894, much better; anasarca about the same, urine 1½% albumin, many casts. January, 1899, dropsy began to disappear; returned to work. May, 1896, feels well; urine 1,029, with a trace of albumin and few casts. April, 1897, excellent health; urine free from albumin and casts; has remained well to this day."

Other physicians who have known cases of chronic nephritis to recover are H. C. Wood and J. P. C. Griffith of

Philadelphia; Beverley Robinson, J. S. Billings and G. L. Peabody of New York; J. B. Herrick of Chicago; E. L. Trudeau of Saranac; and W. S. Thayer of Baltimore.

### "LATENT" CASES.

In addition to our recovered cases there are nine patients who are at work and in good health at the present time, despite the presence of albumin and casts in greater or less quantity in the urine. The duration of these cases, from the first symptoms suggestive of nephritis up to the present time, is as follows:

| | |
|---|---|
| 1 for 6 months. | 1 for 10 years. |
| 2 " 5 years. | 1 " 11 " |
| 1 " 7 " | 1 " 12 " |
| 2 " 8 " | |

Many of these "latent cases" were very striking. The people were able to work hard and steadily for years at a time without interruption, and were entirely free from any disagreeable signs or symptoms, while at the same time the urine contained from a trace to ¼% of albumin and an abundance of casts.

Among the "latent" cases of which notes were sent us by other physicians are the following:

Dr. Franz Pfaff, of Boston, has under observation a patient who has had nephritis for 25 years. The urine shows, as the result of repeated examinations, the following characteristics: amount, 1,680 cu. c.m.; specific gravity, 1,009; albumin very large trace; urea, 1.13%; hundreds of casts in large slide, hyaline, fine granular, coarse granular, a few very highly refracting. Many of the casts are fatty and have renal cells adherent.

Dr. Wharton Sinkler, of Philadelphia, sends the following cases:

No. 1. Mrs. W. had puerperal nephritis and lived 25 years with it. The urine always contained albumin.

No. 2. Mr. E. has had nephritis for about 20 years. It began with an acute attack, with bloody urine. He is still in fair health, though the urine contains albumin.

Dr. Isaac Alder, of New York, sends the following:

"I have under observation a case of chronic parenchymatous nephritis which had lasted for 26 years, and still shows but little disturbances of general health; no edema; no ascites. The urine contains, on the average, two per mille of albumin (Esbach) with granular and fatty casts."

Dr. Alfred Stengel, of Philadelphia, says: "I have under my care two physicians. One has had albumin, casts, and the general symptoms suggestive of chronic interstitial nephritis for ten years to my knowledge. He himself believes that he has had it for 20 years, having discovered albumin in his urine as long ago as that."

"Another case which occurs to me is a lady who has had nephritis certainly for twelve years. Lately, there has been a little swelling of the feet, but this is the only symptom I have ever discovered suggestive of nephritis."

Dr. James Tyson, of Philadelphia, writes:

"Recently I was asked for an opinion in the case of an army officer who had an acknowledged uræmic convulsion 29 years ago; returned to duty, and remained on duty until last summer, when he died of Bright's disease."

## AVERAGE DURATION.

Two hundred and sixty-nine of our cases have died. *The average duration of these fatal cases from start to finish was 19 months.* 40 of our cases lasted less than two months from the first symptom to the time of death. 152 cases lasted two months to two years, and 46 cases from two to five years. The seven longest cases in our series lived 7, 10, 12, 14, 16, 20 and 23 years respectively. Of the fatal cases, 210 were of the type of chronic diffuse nephritis (under which we include those ordinarily classed as chronic

parenchymatous nephritis), and 52 were of the interstitial type.

In the list of 91 cases of long duration sent us by physicians 27 had lasted from 10 to 15 years,

16 " " " 15 to 20 "

6 " " over 20 years, namely, 1 for 22 years.
3 " 25 "
1 " 28 "
1 " 30 "

The number of acute cases in our series was so small that it seemed best to discard them. It is a notable fact, in this connection, that for the last five years, the number of cases diagnosed as acute nephritis at the Massachusetts General Hospital has steadily diminished. We interpret this to mean that cases formerly called acute are now considered as acute exacerbations of chronic cases.

### RELATION BETWEEN ETIOLOGY AND PROGNOSIS.

The nine cases in which there was a distinct history of lead poisoning ran a comparatively long course. The same is true of the 16 cases in which syphilis occurred. Some of the shortest cases, if we are to judge simply by the duration of definite symptoms, were those associated with arteriosclerosis, in which fatal uræmia came on out of a clear sky and killed a patient within a week.

Curiously enough the 57 cases in which the only possible etiological factor obtainable was heredity, that is, a family history of nephritis, "dropsy," apoplexy, heart disease or phthisis, seemed to run a relatively long course.

We were unable to observe any relation between the duration of cases and the occurrence of such etiological factors as abuse of alcohol, infectious diseases, pregnancy, or exposure to cold. *In the vast majority of our cases no cause of any kind could be traced.*

### EFFECT OF COMPLICATIONS UPON PROGNOSIS.

Complications were the cause of death (apparently) in 44 cases out of our 332. Pneumonia and pericarditis were the most frequent complications, and phthisis next. Pneumonia and pericarditis have occurred with about equal frequency, and our statistics indicate that pneumonia is equally common as a cause of death in cases of short and of long duration, while pericarditis seems more apt to carry off a patient at a comparatively early stage of the disease. Thus of the 11 cases ending fatally with acute pneumonia, five cases had a duration of less than two years, and six cases of two years or more. On the other hand, only two of the 15 cases ending with acute pericarditis had a duration of two or more years.

As cases of nephritis get on past the first year the dangers of such complications as cerebral hæmorrhage, hemiplegia and gangrene increase.

### RELATION OF DROPSY TO PROGNOSIS.

It has been said by good authorities that the prognosis in diffuse nephritis is worse in cases where dropsy is marked, but in 47 cases of our series, in which no dropsy occurred, the average duration was 10 months, compared to 23 months, which is the average duration of the cases in which dropsy *was* present. In short, the course of the disease in the cases of diffuse nephritis without dropsy was less than half as long as in the cases with dropsy.

Dropsy is not sufficiently common in interstitial nephritis to be of much prognostic value.

### RELATION OF RETINAL CHANGES TO PROGNOSIS.

Good authorities state that cases of chronic nephritis rarely last more than two years after the presence of hæmorrhages in the retina. This is borne out by our series of cases, of which only one lived over two years from the time at which hæmorrhages were seen in the fundus oculi. In this case,

however, the patient lived five years and seven months after the date at which retinal hæmorrhages in both eyes were observed by Dr. Wadsworth at the Massachusetts General Hospital.

### CARDIAC ENLARGEMENT AND PROGNOSIS.

Occurrence of hypertrophy of the heart is universally acknowledged as marking the advance of the disease beyond the curable stage. This is borne out by our statistics of the 104 cases in which cardiac enlargement was demonstrated. Only three lived more than two years from the date at which the enlargement was demonstrated.

### CONCLUSIONS.

We will summarize our results as follows:

1. Chronic nephritis is not an absolutely incurable disease. Recovery occurs in rare instances.

2. Chronic nephritis may exist for a long series of years without causing any apparent constitutional disturbance.

3. The average duration in 332 cases of chronic nephritis was nineteen months.

4. Acute nephritis is less common than has been supposed, many cases formerly diagnosed as such have been shown to represent exacerbations in chronic nephritis.

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## DISCUSSION.

Dr. R. T. Edes, of Jamaica Plain:—This class of cases is one where there is a great deal of difference of opinion. Those of us who can look back thirty years and can remember all the different forms of kidney disease that were all regarded as Bright's disease can readily see how easy it was to include the kind that we now consider curable with what are now considered incurable. The presence of albumin was then considered a bad sign. That has all been changed. We all admit that we find albumin in conditions that not only improve, but get better. We find more casts

to-day because we probably search more for them than was the case formerly.

My opinion is that casts never come from a healthy tube. It does not of course follow that because one or two tubes are diseased that all the tubes are diseased.

I am astonished that the writers of this paper have found so many cases of recovery. With regard to recovery, I have never seen a case of undoubted nephritis recover. I have seen cases of practical recovery, but never complete recovery.

I remember a case of a woman who has had parenchymatous nephritis for years, where we found plenty of albumin and casts. I have since kept run of the patient, that is, up to a few months ago. That woman is alive and *practically* well. She does not look like a sick woman. If I were to describe that woman's case I would describe it as one where there was still present a certain amount of nephritis.

While I agree pretty closely with what the reader has said, I would not put it in the same words. I would say *practical* recovery.

Dr. E. N. WHITTIER, of Boston:—It ought not to be lost sight of that our associates have reached the same conclusion with reference to duration of life in these cases of Bright's disease as has been reached by one of our large insurance companies with all the wealth of material and resources at its control. After a careful compilation of statistics bearing on rejected risks and subsequent history, the management came to the conclusion that this class of cases was deserving of consideration, and is entitled to some of the benefits of insurance. They have issued what they call a sub-standard policy for this class of applicants. Such cases would all have been permanently rejected a few years ago. The company has come to the conclusion that a mistake has been made in the past by throwing out these cases. They have introduced into this matter of life insurance a new form of policy to cover Bright's disease, etc., and have recognized that it is not alone the healthy public that have rights that insurance companies ought to respect.

Dr. F. W. WHITE:—In answer to the statement that the diagnosis of nephritis 20 or 30 years ago was uncertain,

and thus we may have been led to include cases in our list which were not really nephritis, I wish to say that we have not included any cases of simple albuminuria, that in the selection of these cases of nephritis we have not been guided by the examination of the urine alone, we have also considered the constitutional symptoms, the headache, vomiting and dyspnœa, the cardiac hypertrophy and vascular changes, in short the whole group of signs and symptoms which go to make up the clinical picture of nephritis. With reference to the number of actual recoveries, we are sure of only three cases, and these we have cited. There are probably not more than four or five in all, which does not strike me as a large number of recoveries among 353 cases.

Dr. R. C. CABOT :—In these three cases of recovery there were not any in which characteristic symptoms of chronic nephritis were not present. The three cases which Dr. White has mentioned, I think, were all right.

ARTICLE VIII.

# THE ROLE OF THE STAPHYLOCOCCUS IN SKIN DISEASES.

By CHARLES J. WHITE, M.D.
OF BOSTON.

READ JUNE 13, 1899.

# THE ROLE OF THE STAPHYLOCOCCUS IN SKIN DISEASES.

ONE cannot attend a daily skin clinic without being struck with the frequency with which pus is encountered in the many various diseases which are presented for diagnosis. The conditions where pus is present may be divided into two categories; first, where the disease is primarily and essentially of pustular origin; and second, where pus is a secondary element due to accidental inoculation. In the first division we find impetigo, sycosis (non-tricophytica), ecthyma, furunculosis, abscess and carbuncle; and in the second I mention especially acne, impetiginous eczema, some forms of dermatitis, scabies, certain varieties of iodide and bromide eruptions, and afterwards the primarily vesicular and bullous affections and all cutaneous diseases where pruritus and subsequent scratching play a part. To show a little more precisely how important a place our first division occupies in skin clinics, let me quote a few figures from the returns of the American Dermatological Association, which date from 1877, and of local interest from the records of the skin department of the Massachusetts General Hospital. From the former source we find that the group impetigo, sycosis, ecthyma, furunculosis, abscess and carbuncle constituted 5.19% of all skin diseases recorded, and from the latter records we learn that these same diseases comprised .6% of all the cases observed. As to the precise figures in the secondarily pustular affections it is impossible to speak, as of course there are no such minute statistics in existence.

Suffice it to say that in my investigations the secondarily pustular diseases were exactly twice as numerous as the essentially pustular affections.

Now what is the etiological factor in this important class of dermatoses? Before mentioning the results of any personal work let us see what the more important investigations of others have recorded in this connection, and let me acknowledge here my indebtedness to Unna[1] and to Wickham[2] for many of the extracts which I shall make in the following paragraphs.

In 1877 Vidal[3] made some inoculations from cases of impetigo and concluded his monograph by asserting the possibility of reproducing this disease by inoculation—a fact which, in his mind, established impetigo as a definite dermatological entity. This was, I think, the first experimental work done in connection with this disease. Hebra had never allowed the existence of impetigo as a separate disease, but had considered it a variety of eczema. Tilbury Fox in 1862 had, however, paved the way clinically for the many subsequent investigators, among whom Vidal was the first, as above stated. Three years later, in 1880, Unna[4] produced an essay claiming the inoculability and autoinoculability of impetigo and differentiated it from eczema. In 1881, Crocker[5] found chains of cocci in microscopical specimens from impetigo. In 1885, Eichstedt[6] described an epidemic of impetigo in seventy-six out of seventy-nine children recently vaccinated in Rügen, and considered the affection as a definite one which was inoculable only by intimate contact. This same year Dewévre[7] made a series of inoculations from all products of impetigo and succeeded often in obtaining cultures. Eight years later, in 1893, this same writer[8] produced impetigo artificially by rubbing lice from an impetiginous child upon the skin of a healthy child, and concluded that pediculi were the carriers of the disease—an observation to which many recent writers would agree, although Unna claims merely coincidence.

Bockhart,[9] in 1887, established the extremely important fact—one which Unna characterizes as epoch-making—that from an inoculation from impetigo one could produce impetigo, folliculitis and furunculosis. This assertion has been verified by other observers, and, I think, has been denied by none. Bockhart thus claimed that several diseases, hitherto considered separate and distinct entities, were practically one and the same, that one could pass into another and that the clinical forms were due to a difference in soil, seat and individual and not to a separate etiological factor. Bousquet,[10] two years later, proved that these diseases were due to cocci and reiterated the statements of Bockhart. A few months later Dubreuilh[11]—Bousquet's master—said that "the staphylococcus aureus and perhaps the albus were the pathogenetic organisms," and claimed that there is only one impetigo and that it is inoculable; that furuncles are of the same origin, but lie at a different level of the skin; and that the pustules of scabies are etiologically similar to impetigo and to furunculosis.

In 1890 and 1891 Leloir[12 and 13] described many inoculations from cases of impetigo and found only staphylococcus pyogenes aureus or albus and concluded that all forms of impetigo were inoculable. The following year Leroux[14] continued the work and isolated staphylococcus aureus, albus and citreus and a peculiar form of streptococcus. Dupray[15] spoke only of staphylococci in his thesis in 1891, and some months later Wickham[16] came to similar conclusions in an excellent monograph from which I shall now quote at some length.

Wickham found that on inoculating from impetigo, first from vesicle, then from pustule, then from crust, and finally from excoriation under the crust even after disinfection with alcohol, pure staphylococci resulted each time, and concluded: "first, that staphylococci are found in all secretions of impetigo, in the vesicles as well as in the pustules;

second, that the staphylococci exist in abundance in the inflamed tissues, and that they are in the serous exudations even at the moment of their issue; and third, that each variety of staphylococci can be met with alone or in company with another variety." Wickham inoculated himself on one forearm with the albus and on the other with the aureus. In three days a few miliary pustules appeared which quickly disappeared. He then took a culture of aureus and albus from a non-tricophytic sycosis and one from a purulent zoster and inoculated himself again and found the same results, from which he decided that he did not offer a suitable soil, so he repeated the experiment on a strumous child and produced a typical impetigo contagiosa, which began as an erythema and incipient vesicles within two days and went through the stages of pustulation and crust formation. He then concludes: first, that the inoculation of cultures of staphylococci can reproduce complete impetigo; second, that the inoculation of staphylococci, whether aureus or albus, produces analogous lesions; third, that the staphylococci of impetigo are identical with those of other suppurations, *i.e.*, sycosis, pus of secondary infection; fourth, that certain subjects are rebellious to inoculations and that to succeed one must have great perseverance and irritate the skin violently and apply thick layers of micro-organisms, and fifth, that a lymphatic soil predisposes to impetigo.

In concluding his monograph, Wickham writes: "That certain affections described heretofore as separate diseases—such as impetigo, sycosis non-tricophytica, paronychia, ecthyma, furuncle, folliculitis and carbuncle—are sister maladies, of the same essence; and that the same micro-organism can produce one or another of the aforesaid diseases. Second, that several of these diseases can be found united in the same individual. Third, that therefore none of these affections should be given a special designation, but these diverse affections are not special entities but

only pathological forms of a cutaneous malady, more general, polymorphous par excellence, produced by the staphylococcus and to which it would be suitable to give such a name as staphylococcia purulenta cutanea."

In 1893, Kurth[17] was the first of the more modern investigators to associate streptococci with the name of impetigo. Kurth considered staphylococci only as companions of his streptococcus, which differed in many ways from the streptococcus of Fehleisen. Three years later Brochet[18] published results of a similar nature, but to many his streptococcal theory would appear invalid since re-inoculations from his isolated streptococci produced a lymphangitis and not an impetigo or a folliculitis. Brochet, however, differed from Kurth and others in that he considered his streptococcus to be the same as that of erysipelas, only in an attenuated form.

In 1896, Armstrong[19] observed a series of impetigo cases in Edinburgh football players, and stated that staphylococci were the active agents in the disease. In the following year Balzer and Griffon[20] studied thirty-one cases of impetigo and fourteen of ecthyma, and satisfied themselves that streptococci were the cause of the infections, but re-inoculations produced abscess, erysipelas and fatal septicæmia, and not the original diseases.

Two years ago Curry[21] conducted a series of pathological investigations, and from his results I quote those which apply to the question we are considering. In six pustules he found that four were due to the staphylococcus pyogenes aureus, one to the bacillus of diphtheria, and one to the bacillus pyocyaneus. [I would say that the diphtheritic pustule was due to an accidental inoculation from a case of that disease, and is therefore a case by itself.] In six furuncles, Curry found that four were due to the staphylococcus aureus, one to the streptococcus and one to the bacillus pyocyaneus. In three cases of felon, he found

only the staphylococcus. In three carbuncles a similar result, and in eighteen abscesses he showed that thirteen were due to the aureus, four to the albus and one to the streptococcus. This year Corlett[22] found, on examining nine cases of impetigo, that three were due to the staphylococcus aureus, three to the albus and three to the aureus and albus together.

Thanks to the kindness of Dr. R. M. Pearce, I was allowed to examine the Surgical and Medical Reports[23] of the Pathological Laboratory of the Boston City Hospital, and from them I have selected one case of sycosis, three of furunculosis, two of carbuncle and ninety-five of cutaneous abscess. The six cases of sycosis, furunculosis and carbuncle were due to pure infections of staphylococcus aureus, and in seventy-nine of the ninety-five abscesses, *i.e.* in eighty-three per cent., the staphylococcus is some form was found in a pure state or in conjunction with one or more organisms. To be more precise, I append this table of the analysis of the bacterial examination of the ninety-five abscesses:

In thirty-one cases the staphylococcus aureus was found alone.

In thirteen cases the staphylococcus albus was found alone.

In fourteen cases the two were found in conjunction.

In seven cases the staphylococcus aureus and the streptococcus were found.

In seven cases the staphylococcus albus and the streptococcus were found.

In two cases the staphylococcus aureus and the pneumococcus were found.

In two cases the staphylococcus aureus and a diplococcus were found.

In one case the staphylococcus albus and the bacillus coli communis were found.

In one case the staphylococcus aureus and albus and the streptococcus were found.

In one case the staphylococcus aureus and albus, the streptococcus and the bacillus pyocyaneus.

In fourteen cases the streptococcus in pure cultures was found.

In three cases the pneumococcus in pure cultures was found, and

In one case the bacillis pyocyaneus in pure culture was found.

When Unna published his text-book in 1894 he believed that staphylococci were the cause of impetigo, and said that if we examined the interior of impetigo pustules we should find staphylococci. Now, after a lapse of five years Unna, in collaboration with Frau Schwenter-Trachsler, in a brilliant series of articles, has abandoned his former belief and claims that a special streptococcus is the pathogenetic factor. One point, however, is especially striking, and that is that he no longer searches for the cause in the primary, unbroken pustule, but in the characteristic, exuded, coagulated serum of the impetigo crusts. It is hardly fitting for a young writer to question the verdict of so experienced an authority as Unna, but I should certainly agree with the opinion of Crocker when he says "that search has been made in the crusts (for the cause of impetigo), but their liability to become contaminated is so great that no deductions can be drawn from such observations."

Unna differentiates his impetigo coccus from the streptococcus vulgaris by the following characteristics, which we see are quite unusual: "The chains are always short and composed of from four to six cocci; the chains are quite straight, or at most slightly curved; the chains never form loops or curves; the cocci are irregular and differently shaped; the cocci have their long diameters at different angles; the contents of the cocci often receive the stain unevenly; the colonies are thick and irregular with several short, apparently broken off, branching twigs.

Thus, at too great expense of your patience I fear, I have stated the bacteriological history of impetigo and its sister affections—sycosis, ecthyma, furunculosis, abscess and carbuncle, and I think it is fair to claim that the great majority of investigators concur in the opinion that they are all pro-

bably of staphylococcal origin. There have been a few notable exceptions where men have believed in the streptococcal theory, but to our way of thinking there have always been serious flaws in their arguments.

My own investigations relate to the examination of all pustular lesions which were presented at the skin clinic of the Massachusetts General Hospital between February twenty-third and April thirteenth of this year. The material for inoculation was in all cases taken from under the unbroken skin. In enumerating the diseases, I shall divide them into the two groups which I have described above, and must explain the comparatively small numbers of impetigo and ecthyma by the fact that these studies were carried on during the cooler months when poor children play mostly at home with their arms and legs protected by clothing.

In the first group there were eleven cases of impetigo, ten cases of sycosis, fourteen of furunculosis and two of carbuncle. In the second group there were thirty-nine cases of acne vulgaris, eleven of dermatitis, four of syphilis, three of ringworm, two of lupus vulgaris, two of herpes, two of scabies, two of impetiginous eczema, three of iodide of potash and one of bromide of potash dermatitis, three of dermatitis venenata, one of herpes zoster and one of tuberculous gumma—in other words, one hundred and eleven examinations. From this number the staphylococcus pyogenes aureus or albus appeared eighty-eight times, the bacillus subtilis four times, the micrococcus tetragenus three times, the streptococcus four times, a doubtful bacillus once and in twenty-four instances the tubes remained sterile. From twelve cases there were two different bacteria isolated and in one case three were found. Thus, excluding the sterile cases and the non-pyogenic organisms, we find that in the first division of diseases there were thirty-six infections from staphylococci and one from streptococci, while in the second division there were forty-five from staphylococci and

three from streptococci. Therefore, these Boston figures lend themselves in a very striking manner to the theory of the staphylococcal origin of pustular affections of the skin, and to emphasize my testimony more strikingly I append the following table:

FIRST DIVISION.

| | *Staphylococcus Aureus.* | *Staphylococcus Albus.* | *Streptococcus.* | *Sterile.* | *Uncertain.* |
|---|---|---|---|---|---|
| Impetigo | 7 | 3 | | | |
| Sycosis | 7 | 2 | | | |
| Furuncle | 9 | 2 | | 2 | |
| Carbuncle | 2 | | | | |

FIRST DIVISION (cases with double infection).

| | | | | | |
|---|---|---|---|---|---|
| Impetigo | 2 | 1 | 1 | | |
| Sycosis | 1 | 1 | | | |
| Furuncle | 1 | 1 | | | |

SECOND DIVISION.

| | | | | | |
|---|---|---|---|---|---|
| Acne | 1 | 20 | | 14 | 1 |
| Dermatitis | 4 | 2 | 1 | 1 | |
| K I Dermatitis | | | | 2 | |
| K Br Dermatitis | | | | 1 | |
| Scabies | 1 | 1 | | | |
| Impetiginous Eczema | 2 | | | | |
| Zoster | | 1 | | | |
| Herpes | | | | 1 | |
| Ringworm | | | | 1 | |
| Syphilis | | 3 | | | |
| Lupus Vulgaris | 1 | | | 1 | |
| Dermatitis Medicamentosa | | 1 | | | |
| Dermatitis Venenata | 2 | | | | |

SECOND DIVISION (cases with double infection).

| | | | | | |
|---|---|---|---|---|---|
| Acne | 2 | 2 | | | |
| Dermatitis | 2 | 1 | | | |
| Scrofuloderma | 1 | 1 | | | |
| Herpes | | | 1 | | |

And now in closing let me say a few words about impetigo contagiosa from a clinical standpoint. Etiologically I hope I have made it clear that the disease is in all probability due to the specific action of staphylococci, and in this connec-

tion I want to speak of its far greater prevalence in warm weather.

In the skin department of the Massachusetts General Hospital in 1897 there were one hundred and eighteen cases of this disease recorded; in 1898 there were two hundred and six—a gain of almost one hundred per cent. When we recall the two summers and the two autumns and contrast them one with the other, we shall remember that in 1898 we had an unusually severe summer heat which made itself felt almost into December, while in 1897 we had a comparatively cool summer and an average autumn. Now, if we look at the following table we shall see more strikingly how closely the figures correspond to the severity of the heat.

| | |
|---|---|
| 1897, First Quarter, 21 Cases. | 1898, First Quarter, 34 Cases. |
| " Second Quarter, 14 Cases. | " Second Quarter, 30 Cases. |
| " Third Quarter, 51 Cases. | " Third Quarter, 74 Cases. |
| " Fourth Quarter, 32 Cases. | " Fourth Quarter, 68 Cases. |

There is another etiological point which I wish to bring to your notice, and that is the presence of pediculi on the scalp. I have no direct figures to present in this connection, but you all have noted this coincidence in your practice among the poorer classes. I do not lay too much blame upon pediculi as direct agents in the genesis of impetigo, although I have stated above that Dewévre proved that the disease could be produced by rubbing pediculi from an impetiginous child upon the skin of a healthy child; but I do wish to emphasize the fact that by their presence a scratching of reflex origin is established which causes the child to inoculate himself with the staphylococci which surely abound under his finger nails. The presence of pediculi is therefore a condition upon which we must all strive to make war, especially among school children where the disease is so prevalent. This crusade is now being carried on in the Boston public schools and, I think, with success.

Another point is that in warm weather poor children play about the streets more than in the colder months, and these

same children are apt to wear very little clothing when the heat is intense. Therefore, in summer and in autumn we see impetigo scattered over the whole body, and especially about the face, lower arms and hands, lower legs and feet; while in winter, except in babies, the disease is generally limited to the vicinity of the mouth, nose, ears and eyes.

As to the age at which impetigo contagiosa appears, it is safe to say that nine-tenths of the cases occur in childhood, but there are, however, instances of the affection in adults, and of this latter class I want to say a few words, as it is a condition not quite well enough understood by the general practitioner. In children the clinical picture is quite constant, and we are all well acquainted with the uneven, thin, moist, superficial crusts, varying in color from lemon yellow to orange, which appear, often in the midst of superficial, oozing excoriations upon the more exposed parts of an otherwise rather dirty child. When we come to the adult type, however, we meet a very different condition. Here we find that the subject is almost always a young man, and the seat of the lesions generally the shaven part of the face. We find very often an interesting succession of infections occurring in members of the immediate family or in the patient's intimate companions. The lesions are usually just below the horny layer of the skin and consist of broad, very shallow pustules extending about the shafts of the hair and covered with yellowish-white scales. The progress of the disease is very rapid, and for this reason we are not apt to see isolated lesions, but irregular, gyrate groups covering a considerable area of one or both cheeks. This type is not associated with pediculi, and we seldom if ever find these animal parasites or any evidence of scratching, but we do inquire about the habits of the patient, and often learn that he is shaved at a barber's shop or uses a companion's towel. Here again, therefore, we must enter another plea for more careful cleanliness among both barbers and the public, and

demand a more thorough disinfection of the implements and the utensils of the toilet, both public and private.

As to treatment there are only a few words to say. In the juvenile type plenty of soap and water at frequent intervals, an application twice daily of a five per cent. boracic acid ointment and a thorough soaking of the scalp with crude petroleum for three successive days will, within a week, destroy all active evidences of the disease. We must, however, often wait several weeks for the brilliant erythema, which underlays the crusts, to disappear. In the adult type, on the other hand, we must use rather stronger drugs and must expect a rather longer treatment before the activity of the process is checked. A five per cent. ointment of sulphur, or the application of black wash for fifteen minutes night and morning in conjunction with a five per cent. ointment of salicylic acid and carbolized cosmoline, will usually prove sufficient to cope with the bacteriological invasion. Here again soap and water and clean towels, razors, combs and brushes are indispensable if we wish to be free for now and all time from impetigo contagiosa.

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## DISCUSSION.

Dr. J. C. WHITE, of Boston: I wish to say a word with regard to what investigations of this sort teach us, that a distinction must be made when studying cases of the skin, to separate the essential elements of the disease upon which we make our diagnosis from the changes which are due to secondary infection, which are so very common. Moreover, the treatment in these cases must be greatly modified. We must eliminate the bacteriological elements before we treat the essential part of the disease.

The old methods of studying dermatology have been greatly replaced by work in the laboratory, and the coming dermatologist must pursue many of his investigations in the bacteriological labratory. This is especially true with regard to the study of the most fatal diseases. Bacteriological investigations have assisted us very much in understanding many of these diseases, and the list is being added to day by day.

ARTICLE IX.

# THE IMPORTANCE OF KNOWING THE SIZE OF THE HEART; INACCURACY OF PERCUSSION IN DETERMINING IT AS SHOWN BY X-RAY EXAMINATIONS.

By FRANCIS H. WILLIAMS, M.D.
OF BOSTON.

READ JUNE 13, 1899.

## THE IMPORTANCE OF KNOWING THE SIZE OF THE HEART; INACCURACY OF PERCUSSION IN DETERMINING IT AS SHOWN BY X-RAY EXAMINATIONS.

In the minds of many, cardiac enlargement is chiefly associated with valvular disease, but there are other causes acting outside the heart which may give rise to an increase in the size of this organ. A murmur is to some pathognomonic of heart disease, but this indication is not to be wholly relied upon, for we may of course have murmurs without organic cardiac disease, and cardiac disease without murmurs.

The *size* of the heart, however, is less open to question as a sign of disease, for if this organ is abnormal in size we have an indication of disease either inside or outside of the heart; on the contrary, if the size is normal it is not probable that any serious chronic lesion of the valves is present.

Let me briefly recall to your minds some of the conditions in which the size of the heart departs from that found in health, since the importance of knowing its size may be best appreciated by considering the conditions under which this occurs. I shall exclude from this category the acute changes in the size of the heart which may accompany excessive exercise, or acute diseases such as pneumonia or acute dilatation of the heart. I desire to lay stress upon the importance of knowing when the heart is smaller than normal, for those persons whose hearts are not so large as the body demands should avoid pursuits and exercises beyond their

strength, and to call attention to the fact that with the usual means of examination a small heart is frequently overlooked.

The chief causes of enlarged heart may be divided into two groups: first, those acting from within the heart wall; second, those from without. In this first group, according to Howard, are valvular lesions, and of less frequent occurrence, fatty heart, tuberculosis and aneurism of the heart.

In the second group, causes originating outside of the heart are in the order of their frequency:

Arterio-sclerosis.
Nephritis.
Pericardial adhesions.
Excessive work.
Alcoholic intemperance.
Tumors (pressing on vascular trunks).

Causes involving enlargement of the right side of the heart are obstructions in the pulmonary circulation, and these are found in various pulmonary diseases, especially emphysema.

The recognition of an enlarged heart may not only warn us of the presence of an otherwise unsuspected valvular lesion, arterio-sclerosis, or chronic nephritis, but if its borders are well determined they may also assist us in recognizing the gravity of the conditions causing the enlargement, and careful determinations of the size of the right and left ventricles may aid us much in prognosis. For example, if we find the left side of the heart only slightly enlarged in arterio-sclerosis it is probable that the disease is not so advanced as if the increase in size were greater, nor is the condition so serious as when the right side has also become involved.

Let us now consider the accuracy with which our present methods enable us to determine the size of the heart, and if this consideration shows that they are uncertain and misleading in a matter of such vital importance, we certainly should not rest satisfied with them if other aids can be obtained.

In a considerable number of cases which I have examined during the past three years, I first determined the borders of the heart by percussion and recorded these outlines on the skin with a blue pencil, and then determined the outline of the heart by an x-ray examination and drew these outlines with a black crayon. On the comparison of these two sets of lines I was much struck by their frequent discrepancy in either one or more borders of the heart, and by the greater completeness of the cardiac outlines as obtained by the x-ray examination.

It then occurred to me to test still further the accuracy of the determination by percussion of the right and left borders of the heart by comparing the *width* of the heart as found in the clinical record with the *weight* of the heart as determined by the post-mortem examination. (It is obvious that this method of comparison may err, for a heart may be so distended during life as to give a large cardiac area to percussion, although it may not weigh much more than normal.) With this end in view I attempted to compare the size of the heart in 1,000 patients as found by percussion and given in the clinical records of the Boston City Hospital, with its weight as found in 1,000 consecutive autopsies. For the use of the autopsy records I desire to thank Dr. Councilman, under whose direction the pathological department of the hospital is conducted.

*Four hundred and fifty-four* cases were *not* used for the following reasons:

1. The patients were under 20 years of age.
2. No heart weights were given.
3. The records were incomplete.
4. Cases of emphysema, stoutness, etc., in which percussion is at a great disadvantage.

*Five hundred and forty-six* cases were used.

*One thousand, total.*

The average weights of hearts as determined by Dr. H. D. Arnold at the Boston City Hospital is 290 grams for men and 253 grams for women. I have divided the 546 cases, (370 men and 176 women, the men and women being classed separately) into six groups each, according to their weights, as will be seen in the following tables, marked *A*.

*A*

HEART WEIGHTS COMPARED WITH HEART WIDTHS.

*MEN. Average weight in health 290 grams.*

*Group I.*—Weights below 225 grams. Nine cases.
7, area normal by percussion.
1, area enlarged by percussion. Small size not recognized in 88%.

*Group II.*—Weights 225–349 grams. One hundred and sixty-eight cases.
11, area diminished by percussion. Error of 6%.

*Group III.*—Weights 350–399 grams. Seventy-four cases.
58, area normal by percussion.
6, area diminished by percussion. No enlargement recognized in 83%.

*Group IV.*—Weights 400–449 grams. Twenty-six cases.
15, area normal by percussion.
1, area diminished by percussion. No enlargement recognized in 61%.

*Group V.*—Weights 450–499 grams. Twenty-four cases.
9, area normal by percussion. No enlargement recognized in 37%.

*Group VI.*—Weights 500 grams and over. Sixty-nine cases.
18, area normal by percussion.
1, area diminished by percussion. No enlargement recognized in 27%.

*A*

HEART WEIGHTS COMPARED WITH HEART WIDTHS.

*WOMEN. Average weight in health 253 grams.*

*Group I.*—Weights below 200 grams. Eight cases.
7, area normal by percussion. Small size not recognized in 87%.

*Group II.*—Weights 200–324 grams. Ninety-nine cases.
*12, area enlarged by percussion.

*Group III.*—Weights 325–374 grams. Twenty-six cases.
17, area normal by percussion.
1, area diminished by percussion. No enlargement recognized in 69%.

*Group IV.*—Weights 375–424 grams. Fifteen cases.
5, area normal by percussion.
2, area diminished by percussion. No enlargement recognized in 46%.

*The weights of these twelve hearts were as follows: One weighed 230 grams; two, 245; one, 250; one, 260; one, 270; one, 280; four, 300; one, 315.

*Group V.*—Weights 425–474 grams. Twelve cases.
4, area normal by percussion.
1, area diminished by percussion. No enlargement recognized in 41%.

*Group VI.*—Weights 475 grams and over. Sixteen cases.
1, area normal by percussion.
1, area diminished by percussion. No enlargement recognized in 12%.

If you will follow these tables you will see that this comparison between the *weight* of the heart as determined at the autopsy and the *width* of the heart as obtained by percussion shows that:

Group I.—Men.—In nine men with hearts weighing below 225 grams (average weight of normal heart 290 grams) the diminished size was not recognized by percussion in 88% of the cases. The number of cases in this group is small, but in the corresponding Group I. of the women the results are similar.

Group II. In 168 men with hearts varying in weight from 225–349 grams, a variation which I have allowed as normal either side of the 290 grams, 6% were diminished in size by percussion. The weights of those included in this percentage were:

| | | | |
|---|---|---|---|
| 1 | weighed | 250 | grams. |
| 1 | " | 280 | " |
| 2 | " | 290 | " |
| 1 | " | 300 | " |
| 2 | " | 310 | " |
| 1 | " | 320 | " |
| 1 | " | 325 | " |
| 1 | " | 330 | " |
| 1 | " | 340 | " |

Group III. In 74 men with hearts weighing from 350–399 grams (average weight of normal heart 290 grams) no enlargement was recognized by percussion in 83% of the cases, and six indeed were even recorded as smaller by this means of examination.

Group IV. In 26 men with hearts weighing from 400–449 grams (average weight of normal heart 290 grams) no enlargement was recognized by percussion in 61% of the cases, and as in the previous group one was diminished.

Group V. In 24 men with hearts weighing from 450–499 grams (average weight of normal heart 290 grams) no enlargement was recognized in 37% of the cases.

Group VI. In 69 men with hearts weighing 500 grams and over (average weight of normal heart 290 grams), no enlargement was recognized by percussion in 27% of the cases and one was recorded as diminished in size by this method.

It is not necessary to go over the table for women, as it is of similar import.

In this comparison I have given the clinical record the benefit of all doubt; for example, I have excluded all cases of emphysema and stoutness.

It is worthy of notice that both these tables show that when the heart is about normal the error made by percussion is least frequent; when smaller than normal, very frequent; when somewhat enlarged, nearly as frequent; as the heart grows larger and larger, the frequency of the error made by percussion decreases, but the amount of error in a given case becomes greater.

Let us now return to Group IV. for a moment. In this group of twenty-six men with hearts weighing from 400–449 grams, no enlargement was found by percussion in sixteen of the cases; in only two of these were there murmurs;* in the remaining ten, which were recognized as enlarged by percussion, four had murmurs.

In Group V., twenty-four men with hearts weighing from 450–499 grams, nine hearts were given as normal by percussion; in only two of these were there murmurs; in the remaining fifteen, which were recognized as enlarged by percussion, eight had murmurs. This may mean that when murmurs are found closer attention is given to the size of the heart.

* At the meeting, tables were presented which showed that in certain of the 546 hearts under discussion murmurs were present both when the valves and weights were normal and abnormal, but these tables need a fuller discussion than the limits of this paper warrant, and I have therefore not printed them.

It is evident from the comparison we have made between the size of the heart as obtained by an autopsy and by percussion that the latter method is inaccurate. Now let us return to x-ray examinations and see how percussion stands as to accuracy when compared with a method which, like percussion, is used during life. (The determination of the right and left borders of the heart was made on a level with the nipples in this comparison.) It is unnecessary to compare the two methods of determining the outlines of the heart as far as completeness goes; the x-ray examination gives much fuller and more detailed outlines.

I will ask your attention to the table marked *B*, which gives the results of this comparison in 107 cases of various diseases. These cases are only a part of those that I have examined with the fluorescent screen, but are those in which I have employed the two methods, *percussion* and *x-ray examination*. The left border in these 107 cases was determined by its distance from the median line in both methods of examination. The figures give the difference in the distance of the left side of the heart from the median line as determined by percussion and x-ray examination in each patient. The plus mark means that percussion made the heart larger than did the x-ray examination, and the minus mark that percussion made the heart smaller.

The table consists of four double columns; in the first column, thirty-one cases, in one-third of which the hearts were larger than normal, the difference between the left border as determined by percussion and x-ray examination was less than one centimetre. This we will call agreement.

In the second column, thirty-seven cases, in one-half of which the hearts were larger than normal, the difference was one centimetre or more, and less than two.

In the third column, twenty-five cases, in one half of which the hearts were larger than normal, the difference was more than two centimetres and less than three.

In the fourth column, fourteen cases, in one-half of which the hearts were larger than normal, the difference was more than three centimetres.

The table also shows whether the disagreement between the two methods was more often plus or minus in the 107 cases. If we deduct from the total number the thirty-one cases, in which I have called the results of the two alike, we have left seventy-six cases. By percussion thirty-nine of these were larger and thirty-seven were smaller than by an x-ray examination.

In the cases in the left hand side of each double column the total width of the heart by an x-ray examination was thirteen centimetres or less; in those in the right hand side it was over thirteen centimetres. (The average width of the heart in health as determined by an x-ray examination in forty-eight men was, from the median line to right border, 3 cm., to left border 8.6 cm., total 11.6 cm.; in thirty-one women the right border was 2.5 cm., left border 8.7 cm., total 11.2 cm. These cases indicate that the heart in women lies a little more to the left than in men.) Thus it will be seen that in cardiac disease the error made by percussion was more frequent among the larger hearts than among those of nearly normal size.

It should be stated that I have taken pains, from time to time, to get other physicians to determine by percussion the size of the heart in some cases, and I then compared it with that obtained by an x-ray examination; as yet I have found no one whose percussion like mine in the same cases did not give the size of the heart incorrectly when tested by an x-ray examination.

If we consider the table (B) in a general way and look at the groups of diseases, it seems that percussion is very likely to err in finding the position of the left border of the heart when it is of the most importance to have accurate information about this organ, namely, in cardiac disease

*B*

## Difference of X-Ray and Percussion Determinations of Left Heart Border in 107 Cases.*

| Less than 1 cm. | 1–2 cm. | 2–3 cm. | 3 cm. and over. |
|---|---|---|---|
| *Aneurism.* | *Aneurism.* | *Aneurism.* | *Aneurism.* |
| 0 | +1.75 | +2.50 +2.00 | +3.00 −4.50 |
| 0 | | +2.00 | |
| | | | *New Growth.* |
| | *Bronchitis.* | *Bronchitis.* | −4.25 |
| | −1.00 | +2.00 | |
| | +1.50 | | |
| *Cardiac.* | *Cardiac.* | *Cardiac.* | *Cardiac.* |
| 0 +.50 | +1.00 −1.00 | −2.00 +2.00 | −3.00 |
| 0 | −1.00 +1.00 | +2.50 −2.00 | +3.00 |
| 0 | +1.25 −1.00 | +2.50 −2.00 | +4.00 |
| 0 | −1.50 −1.00 | +2.75 −2.00 | |
| 0 | +1.75 +1.00 | −2.25 | |
| | +1.75 −1.25 | −2.50 | |
| | −1.50 | −2.50 | |
| | −1.50 | −2.50 | |
| | −1.75 | | |
| | *Nephritis.* | | |
| | +1.00 | | |
| *Anæmia.* | | *Anæmia.* | |
| +.25 | | −2.00 | |
| +.75 | | | |
| 0 | | | |
| *Rheumatism.* | *Rheumatism.* | *Rheumatism.* | |
| −.50 | +1.25 +1.50 | −2.25 | |
| +.75 | +1.75 | | |
| *Pleurisy.* | *Pleurisy.* | | |
| +.50 | −1.75 −1.00 | | |
| | −1.00 | | |
| *Tuberculosis.* | *Tuberculosis.* | *Tuberculosis.* | *Tuberculosis.* |
| 0 −.50 | +1.00 +1.50 | +2.00 +2.50 | +3.00 +3.50 |
| +.25 | +1.00 −1.75 | −2.00 | −3.25 −4.25 |
| −.25 | +1.25 | +2.50 | +4.00 |
| +.50 | −1.25 | | −4.00 |
| −.50 | | | |
| −.50 | | | |
| −.75 | | | |
| −.75 | | | |
| *Pneumonia.* | *Pneumonia.* | *Pneumonia.* | *Pneumonia.* |
| 0 −.25 | +1.00 +1.75 | −2.50 | −4.00 |
| −.25 −.25 | −1.00 +1.75 | | |
| −.25 −.50 | −1.50 | | |
| *Typhoid Fever* | *Typhoid Fever.* | | |
| −.50 | +1.25 | | *Pericardial Adhes.* |
| | | | −4.75 |
| *Tape Worm.* | | | |
| −.50 | | *Emphysema.* | |
| | | +2.25 | |
| | | *Malaria.* | |
| | | +2.00 | |
| Total, 31 | Total, 37 | Total, 25 | Total, 14 |

* + Means percussion made the heart larger than the x-ray examination.
− Means percussion made the heart smaller than the x ray examination.
The left hand column in each of the four double columns contains the cases where the heart's width was 13 cm. or less; the right side those in which it was over 13 cm.

when the heart is enlarged. Percussion also errs when the heart is displaced. To illustrate the advantage of an x-ray examination in this latter case, let me refer briefly to a patient who consulted me recently for supposed cardiac disease. The left border of the heart, as well as the apex beat, was outside of the nipple line; the right border was not easily defined, but seemed to be in about the usual place. Without an x-ray examination I should have considered that he had an enlarged heart; this examination showed, however, that the heart was not enlarged but was simply drawn to the left. Upon inquiry I found that he had had a left-sided pneumonia forty years before. When the heart is pushed out of place by fluid or a tumor in one or both sides of the chest or in the abdomen, the displacement as a rule is much more certainly recognized by an x-ray examination than by other means.

In the few cases in which I have had an opportunity to compare the size and position of the heart, as shown by an x-ray examination, with that found at autopsies, they have all agreed; except that after death the heart was somewhat higher than in life.

When the heart is normal in size, its border, so far as it can be obtained by percussion, can usually be obtained correctly; when it is of abnormal size, whether smaller or greater, this condition may not be indicated by percussion.

If by an x-ray examination we find in any patient (excluding acute diseases) an enlarged heart we should consider the probability of valvular lesions, arterio-sclerosis (especially in middle age and after), renal disease and emphysema (this last condition of the lungs would be very obvious by an x-ray examination).

If we find the heart normal in size and no history of disease standing in causal relation to valvular trouble, we probably have not to do with a serious chronic condition of the heart even though some murmurs are present.

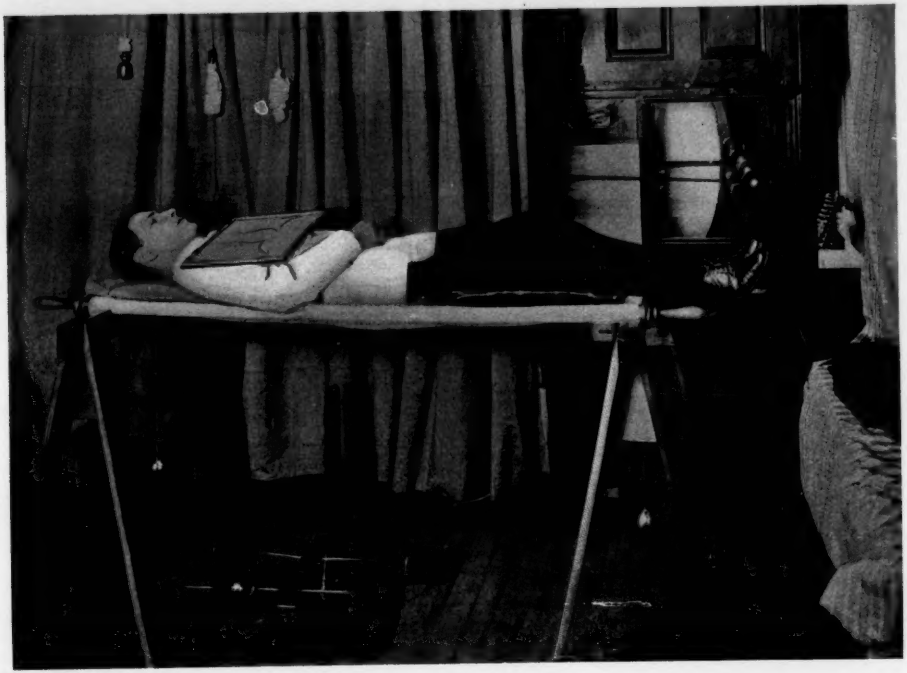

Fig. I, A.

Small hearts may be recognized by an x-ray examination.

An x-ray examination, when the lungs are clear,* enables us to distinguish the outlines of the ventricles more exactly than percussion; a displaced heart is not mistaken for an enlarged one; the direction of the long axis can be obtained; if the heart is abnormal in size we can ascertain whether the right or the left or both ventricles are enlarged; the enlargement of the auricles can be distinguished.

In a word the size of the heart can be determined more accurately and more certainly by x-ray examinations than by our former methods; these examinations are of especial value in the early stages of cardiac enlargement as then so much more can be done for the patient than when the enlargement has made great progress.

Fig. 1, A. This cut shows my method of making x-ray examinations of the thorax. The patient is lying on a stretcher and the anode of the vacuum tube is under the point where the median line is crossed by a line joining the nipples and three or four feet from it. The fluorescent screen (covered with a transparent film on which the cardiac outlines have been drawn) lies on the patient's chest. One of the cords over the patient's head is used to turn the electric current on or off; the two others are used to increase or diminish the light as may be desired while the examination is being made. The aluminium screen, which is always placed between the Crookes tube and the patient, has been taken away in order to show the position of the tube. The curtain is drawn aside at the foot of the stretcher in order to show

* Unless the thorax is too opaque to the x-rays from disease we see a portion of the left border of the heart clearly defined during quiet breathing; we do not, however, always see the right border at this time. During a deep inspiration the whole of the left border of the heart is clearly seen and the right border also comes into view. The width of the heart can therefore be best obtained during deep inspiration, as then both borders are seen together; but there may be an error in some cases in this determination on account of the heart's being compressed by the lungs In cases where the heart is enlarged so that both borders may be seen at the same time during quiet breathing, its width may then be determined; the diminution in width during full inspiration as compared with quiet breathing may sometimes aid us in distinguishing a heart that is dilated from one that is hypertrophied.

the end of the case which encloses the static machine. This machine has four revolving plates six feet in diameter, and four fixed plates six feet four inches in diameter and adjustments requisite for examinations of the thorax. (See Fig. 1, B.)

If the heart is enlarged each border may be determined separately by the aid of an indirect plumb line which I devised for this purpose.*

I desire to express again my obligation to Dr. William H. Rollins of Boston, not only for the advances which have resulted from his studies of vacuum tubes, by which I have been greatly aided, but also for his constant helpfulness in furthering my work.

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## EXPLANATION OF HEART OUTLINES.

Figures 2–7 inclusive are photographic reproductions two-thirds the size of the original tracings made on the chest wall to show the position of the heart's border as determined by percussion (broken lines) and by x-ray examination (full lines).

In Fig. 2, John W. M., the x-ray line of the left border of the heart was outside of the percussion line.

In Fig. 3, Catherine P., the position of the left border of the heart as determined by the x-ray examination was inside of the line as indicated by percussion.

In Fig. 4, Mary F. D., the right chest contained fluid which pushed the heart to the left; this displacement was not recognized by percussion. It may be that displacements are sometimes not readily recognized by percussion, because the heart is pushed into the body of the lung rather than along or near the chest wall.

Fig. 5. Mary M. This was an unusual case. There was fluid in the left chest which displaced the heart to the right, but as she had also at the same time pneumonia in the lower part of the right lung the heart was not pushed directly to the right, but was forced upwards. Hence the right border of the heart was found to the right and above a line joining the sternal notch and the right nipple.

* Amer. Jour. Med. Sciences, June, 1899, p. 688.

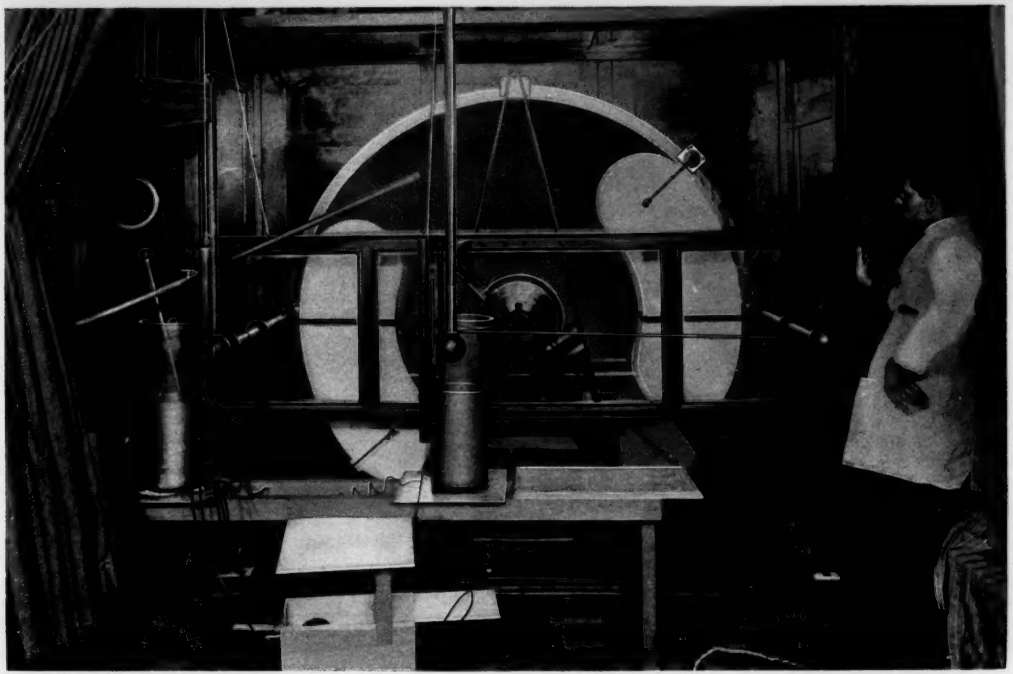
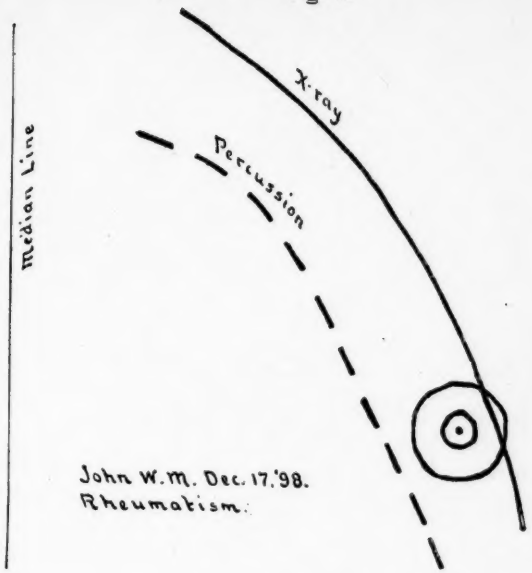


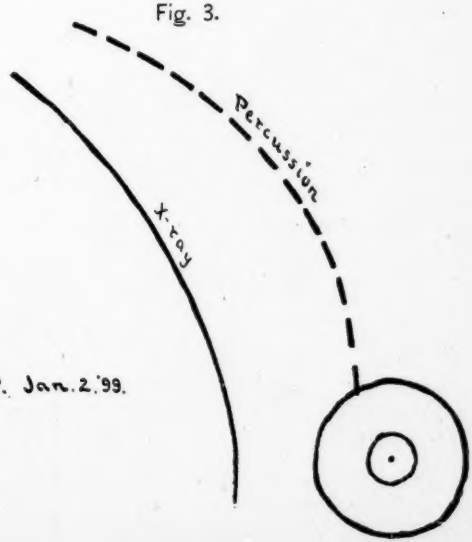
Fig. I, B.

Fig. 2.



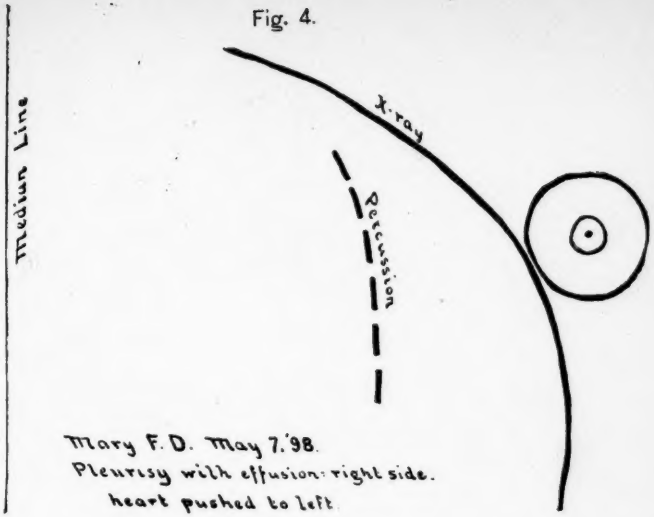

John W.M. Dec. 17.'98.
Rheumatism.

Fig. 3.



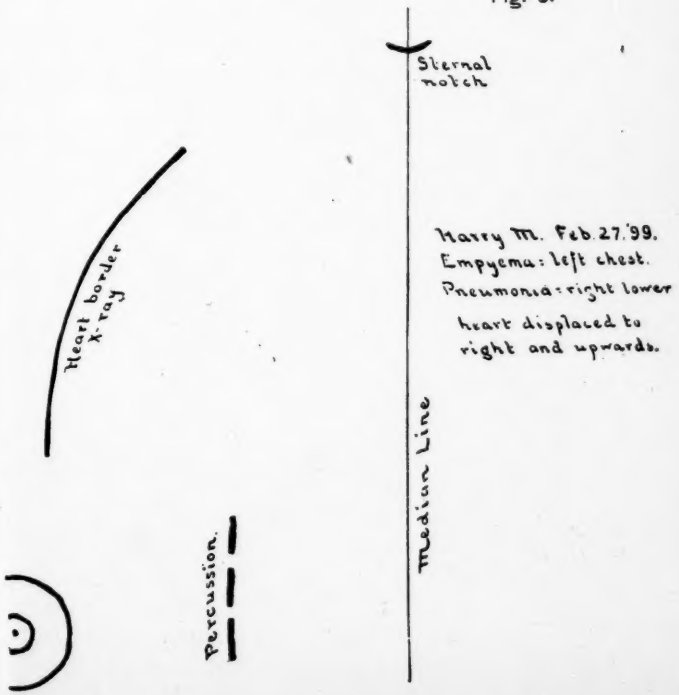

Catherine P. Jan. 2.'99.
Hysteria.

Fig. 4.



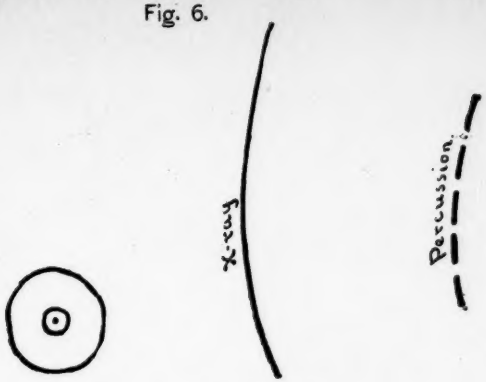

Mary F. D. May 7.'98.
Pleurisy with effusion: right side.
heart pushed to left

Fig. 5.



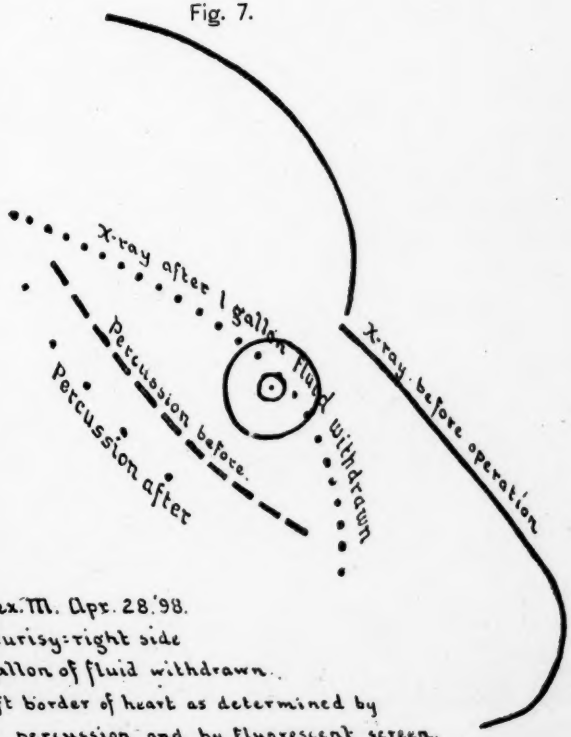

Harry M. Feb 27.'99.
Empyema: left chest.
Pneumonia: right lower
heart displaced to right and upwards.

Fig. 6.




Constantin D. Jan. 23, '99.
Pleurisy with effusion left side;
heart pushed to right.

Fig. 7.




Alex. M. Apr. 28. '98.
Pleurisy: right side
1 gallon of fluid withdrawn.
Left border of heart as determined by
percussion and by fluorescent screen.

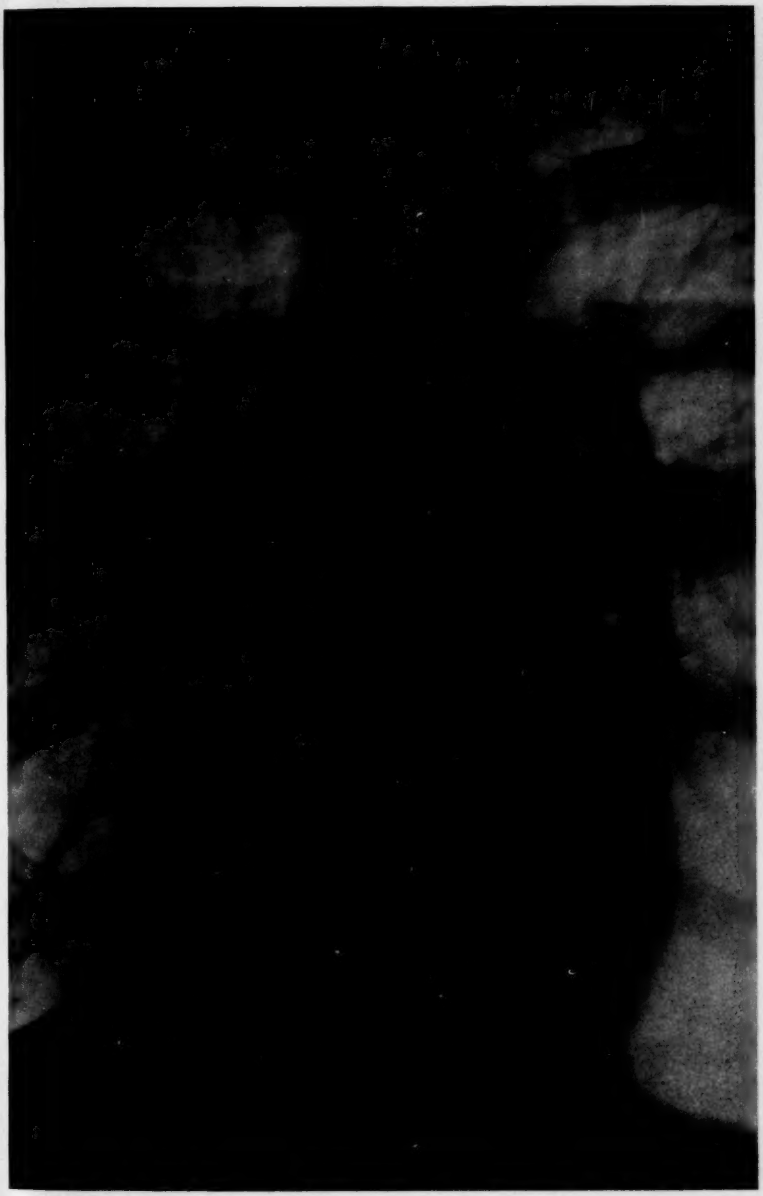


Fig 8

Fig. 6. Constantin D., shows the right nipple and the right border of the heart pushed towards it by fluid in the left chest. In this patient percussion did not recognize the right border of the heart within an inch and seven-eighths.

Fig. 7. Alex. M. had a very large pleuritic effusion in the right side which pushed the heart far to the left and much outside the left nipple. The position of the left border of the heart before the fluid was withdrawn is shown by the full line, after its withdrawal by the dotted line crossing the nipple. The position of the same border as determined by percussion before the operation is shown by the broken line, after operation by the dotted line below it.

During quiet breathing only a portion of the heart's border is visible by an x-ray examination, but as I pointed out in 1896, the patient should take a full breath when we wish to see the outlines of the heart more fully. Thus far I believe the heart has never been photographed between the respirations and during full inspiration. Figure 8 is a reproduction of an x-ray photograph of the heart taken under these conditions. It is about one half the original size and shows, above the fourth ribs, the outer border of the superior vena cava and of the aorta, below the fourth ribs, the heart; the diaphragm is seen at the bottom of the picture.

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## DISCUSSION.

Dr. F. I. KNIGHT, of Boston: I do not feel able to criticise the main part of this paper. We all agree that to be able to determine the exact size of the heart is very important. There are certain conditions in the chest which interfere very much with determining the exact size of the heart by percussion, principally emphysema. The x-ray photograph in this instance is of great assistance. Enlargement of the liver, certain conditions of the stomach, the distortions produced by pleurisy with effusion, offer obstacles to our efforts to determine the exact size of the heart by percussion.

The future, however, must decide the exact value of the x-ray with reference to its ability to tell us the exact size of an organ.

I remember one source of error regarding the x-ray in this respect. In this instance the percussion outline was a good deal larger than the outline made by the x-ray. This was a case seen by Dr. Williams and myself. When I called his attention to this discrepancy Dr. Williams diminished the amount of light, and the result was the same outline that I got by percussion.

Again, by the use of the x-ray we may be able to ascertain whether the walls of the heart are or are not very thin.

Dr. F. H. WILLIAMS: What I have shown this afternoon is only a beginning in this kind of work, and the percentage of cases in which percussion is in error and the amount of this error is, of course, not fully determined. There are sources of error, as Dr. Knight has said, in making x-ray examinations, if proper precautions are not taken, and I am glad that this point has been brought up. The light, for example, must be just right. The case referred to by Dr. Knight was a young boy, and the light was too strong, having been adjusted for the previous patient, who was an adult; my interest at that moment was directed to showing Dr. Knight my method of examination rather than to the making of the examination itself. In examining young patients, where the heart is naturally small, if there is too much light, there may be no shadow or almost no shadow of the heart cast on the fluorescent screen; or the heart may seem to be diminished in size because its edges have been penetrated by the rays. I have insisted for fully three years that a means for adjusting the light while using the fluorescent screen is essential, just as an adjustment of the light is necessary for the microscope, and I have devised a simple contrivance which enables me to vary the light while making an x-ray examination; one hand should always be on the adjustment which controls the amount of light so that it may be varied according to the part which is being studied, or the size, or age of the patient. If this precaution is taken the best light can be obtained if the apparatus is efficient and the physician is experienced. The ability to see the size and position of the heart is unquestionably an advance upon our other methods of examining it.

ARTICLE X.

# INTUSSUSCEPTION.

By JOHN C. MUNRO, M.D.
OF BOSTON.

READ JUNE 13, 1899.

# INTUSSUSCEPTION.

THAT intussusception is not rare is shown by the fact that over five hundred cases have been reported within the last twenty-five years. According to Leichtenstein forty per cent. of all cases of intestinal obstruction are due to this lesion. Probably three-fourths of the cases occur in infants, though no age is exempt, even the fœtus at full term not escaping. The lesion is due to the swallowing by one portion of the gut of its neighboring piece which has become constricted in consequence of a vigorous contraction of its circular fibres. According to Power, children are predisposed apparently on account of the greater length of the mesentery.

All cases should be considered, in the interest of the patient, to be surgical from the beginning. A strangulated hernia has long since ceased to be a medical disease; how much more should an internal strangulation with its similar possibilities be within reach of surgical interference if necessary. As in appendicitis, every case may not demand operation, but the responsibility should be placed in the hands of one who is ready and able to operate at the earliest opportunity.

Intussusception may be caused by sudden blows or injuries, muscular efforts, tumors of various kinds, typhoid or tubercular ulcerations, diarrhœa and possibly vomiting and coughing.

The invagination is found in more than half of the cases in the ileocæcal region, though other portions of the in-

testinal tract may be involved, Chiari and Engel each having seen a partial invagination in the stomach. The jejunum is comparatively rarely affected.

The symptoms in a typical case are sudden and acute abdominal pain, usually coming on at night, vomiting and stools containing mucus and blood. There may be diarrhœa or obstipation. A tumor, not necessarily sausage-shaped, can be felt in the abdomen, generally on the right side, but until peritonitis has begun, spasm is absent or slight in degree. As the intestine becomes more and more constricted, congestion, stasis, infiltration and necrosis follow. Owing to the greater elasticity of the constricting ring as compared with the ring in external hernia, we are less liable, comparatively speaking, to find strangulation. Furthermore, the inner tubes being more liable to become gangrenous than the sheath, peritonitis may be escaped by the passage of the gangrenous portion along the digestive tract. But, in actual practice, it is not safe to rely on so slight an advantage, or to consider the condition less serious than it is, to wit, one of the most serious that can confront the surgeon.

Intussusception may be acute, chronic, chronic with acute exacerbations; single or multiple; direct or reversed.

Peritonitic adhesions may not form for weeks, or may begin with the onset of the invagination.

Spontaneous recovery may take place by disinvagination or by separation of the inner tube and evacuation later; in the latter way several feet of gut may be lost, one observer (Peacock) having seen eight pieces, aggregating twelve feet, passed during the course of three years. Disinvagination without interference beyond medical treatment occasionally takes place, but such a fortunate outcome is rarely to be anticipated. Of spontaneous separation Rydygier says that it is very satisfactory but rare, "and to speculate on a chance accident is not an established therapeutic procedure." Many

such cases die later of perforation, peritonitis, stricture, etc. According to Senn, young children and infants seldom, if ever, recover in this way.

Treatment by mechanical distension with liquids, air or gas, may be successful if employed early, that is within the first twenty-four or forty-eight hours at the utmost. In acute cases it is best given with full anæsthesia and the patient inverted, all preparations for laparotomy in case of failure, necessity or doubt having been made. The pressure should be slight, not over two pounds if gas be used, or if a liquid, the reservoir should not be over two or three feet above the patient. A low intussusception may rarely be pushed back by a finger or tube passed into the rectum.

There is considerable shock from such a simple procedure, and many cases of rupture of the intestine during injection have been recorded. It is difficult to reduce the final inch of the invagination, and without actual inspection one cannot tell whether the reduction has been complete or not, so that the cases of recurring intussusception are probably due to the failure of complete disinvagination and not to a fresh, independent attack. Barker says that there is a tendency of the bowel to unroll itself *up to* the point at which it first began, but no farther; in other words, the supposed reduction is not a complete one, and abdominal palpation under such conditions may fail to show any tumor.

In brief, injection is justifiable in chronic cases; where the patient refuses operation in acute cases; when beyond surgical aid and within a few hours after the onset of the attack. If the first attempt in acute cases is not successful it is useless to make a second trial.

All authorities urge operation in one form or another, as early as possible in acute cases and at once after trying a simpler procedure such as injection, for the same reason that one should be prepared to attack a hernia after failure to reduce it by taxis. In chronic cases it is unwise to delay

over a week, simple attempts at reduction having been made meanwhile. The patient should be watched closely to forestall any acute attack. As in operating on the appendix in the interval the risk of a laparotomy is reduced to its minimum.

After opening the abdomen there are various methods of dealing with the many conditions found. Of these we may consider artificial anus, entero-anastomosis, disinvagination or reduction, complete and partial resection.

Artificial anus has very little to recommend it. There is scarcely a recorded recovery in an acute case. Occasionally a chronic case has survived, and an acute case where a resection has followed the artificial anus which was resorted to to tide the patient along temporarily. As it does not relieve the strangulation or its consequences except the distension and obstruction it must be looked upon merely as a makeshift or an evidence of faintheartedness.

Entero-anastomosis has very little more to recommend it. The intestinal current is, to be sure, re-established, but the invagination with all its possible and probable consequences still remains. A few cases, less than a dozen, of recovery in adults have been reported, most, if not all of them, being in chronic intussusception.

Disinvagination or reduction is the ideal operation, and if the case is seen early, before necrosis and firm adhesions have taken place, the outlook for its successful employment is good. Everything depends on the interference being early. Fifteen years ago Treves found that the mortality was only 30% in cases easily reduced, as against 91% in those reduced with difficulty, and since then, with the improvement in technique and diagnosis the early operation shows a much lower death rate.

Where the reduction is not easy on account of firm adhesions there is always a risk of tearing the bowel or of gangrene from the prolonged pressure, and in such suspicious cases it is safer to isolate or drain the area involved. Chronic

cases are not necessarily difficult to reduce, even after weeks or months of invagination. In reducing, Senn advises that the œdema be treated first as in a phimosis and then that counter-traction be made below the apex in order to prevent folding, in the sheath and middle cylinders; occasionally insufflation will add materially in the manipulation. To prevent re-invagination if it appears liable to take place, he also suggests that the mesentery be shortened.

Where, owing to adhesions, tumors, necrosis or perforation, disinvagination is not possible, a resection in one form or another must be resorted to. Probably the simplest method is that devised by Maunsell or some modification similar to that described by Rydygier. The entering gut is sutured to the sheath at the neck without disturbing its position, and then through a longitudinal incision in the sheath the invagination is delivered and amputated near the neck, the free edges of the stump being sutured together, controlling in that way the mesenteric vessels. After dropping the stump back into place, the opening in the sheath is closed. If the invagination cannot be delivered through the side opening after amputation it may be left to come away later in the stools. Where possible, a typical Maunsell operation may be done. In this way the least possible amount of intestine is sacrificed and the mesentery is quickly and easily controlled. The use of buttons or spools is unnecessary.

Where the sheath is gangrenous a complete excision is enforced, end to end or lateral anastomosis being employed. Here plates or buttons may be used if thought best. Occasionally it may be found that all of the intussusceptum and a portion only of the sheath may be removed. If the intussusception is low down re-section may at times be done through the rectum without opening the abdomen.

As, for the most part, all data and methods are based on intussusception in the child and in the ileo-cæcal portion of

the intestine, it may be well, in view of the two personal cases reported to-day, to consider, in more detail, invagination in adults and especially when it takes place in the jejunum.

Adding to Braun's table made in 1885 and to Rydygier's table made in 1895, fourteen cases of reduction or re-section in adults over sixteen years of age, reported since, we have a group of seventy-two cases altogether. Undoubtedly there have been many more cases that have been operated upon, but so far as could be discovered no more have been recorded.

Of twenty-one recoveries after reduction—including the cases of Perceval (1888), Stretton (1894) and Barker (1897)—thirteen were in the ileo-cæcal or colic region, four were iliac, one jejunal and three unknown. Thirteen were chronic cases, seven acute, and one unknown.

Of thirteen fatal reductions—including the cases of Davies-Colley, White (1889), Richardson (1892)—nine were ileo-colic, one each, iliac and jejunal and two unknown. Seven were acute and six were chronic, probably some of the latter being operated upon during an acute exacerbation. Shock and peritonitis were the main causes of fatal issue.

Of seventeen recoveries after re-section—including the cases of Hartley (1892), Wassiljew and Munro (1898)—fourteen were ileo-colic and one each were iliac, colic and jejunal. Six were acute or chronic with acute exacerbations, and nine were chronic, while two were unknown. In two cases (Mikulicz and Wassiljew) the re-section was secondary to an artificial anus.

Of twenty-one fatal re-sections—including the cases of Abbe (1891), Senn, Robson and Munro (1897)—nine were ileo-colic, seven iliac, one colic, two jejunal and two unknown. Fourteen were acute or chronic with acute exacerbations, five were chronic and two unknown. One (Esmarch) was secondary to artificial anus. Nearly all died of peritonitis or shock.

The jejunum was involved in only five of these cases.

1. White's case, an acute invagination ten inches long and two and one-half feet from the pylorus, died on the table, the operation being abandoned.

2. Stutsgaard's case, caused by a lipoma and of unknown duration, died in five days of peritonitis after re-section of 128 cm.

3. Robson's case of seven days' duration, died after re-section of four feet and an end to end anastomosis.

4. Borch's case of acute ileo-jejunal invagination recovered after disinvagination.

5. Writer's case of recovery after re-section of thirty-two inches of jejunum.

The earlier cases, like those reported by Velse, Fuchsius and others at the beginning of the present century were naturally cases of reduction, many of them being easily replaced. When in recent years re-section began to be employed it was at first almost uniformly fatal, but with the improvement in technique the mortality has fallen nearly one-half, and in all probability it will never be much less because, with earlier diagnosis and operation, re-section will be replaced by reduction, leaving only the desperate and neglected cases for the more serious operation.

Where the lesion involves the upper portion of the intestine any procedure must of necessity be fraught with more danger than in the cases in which the lower part of the tract is involved, but fortunately the latter region is far more frequently affected, so that with early surgical supervision and early interference in both acute and chronic cases after a judicious trial of simpler, non-operative means, many more patients should be saved. It remains in the hands of the general practitioner to realize the grave possibilities in all cases and to protect himself and his patients by early resort to the surgeon, who, on his part, must appreciate that death, just as in strangulated hernia or in acute appendicitis, is too often the price of a useless delay.

As a contribution to the statistics of operation for intussusception in adults, the writer will add two cases of his own. Both have been briefly reported in the Boston City Hospital Medical and Surgical Reports, Series X., but it may be allowed to give the history of one a little more in detail, the fatal one being dismissed in a few words.

Case 1. Male, thirty-seven years old, was admitted to Dr. H. W. Cushing's service at the Boston City Hospital with a history of abdominal pain, obstipation, distension and tenderness in the region of the appendix for sixty hours. Operation under ether. Intussusception of the lower end of the ileum eight inches long and with beginning gangrene was found. Amputation proximally seven inches from the invagination and distally close to the cæcum, or about one inch from the invagination with end to end anastomosis was made. Patient in poor condition at time of operation. On the following day he was still very ill and got out of bed against orders. Sixty hours after operation he died, a partial autopsy showing the abdomen to be full of fæces from a rupture in the intestine on the proximal side of the joint, which was tight.

The history of the case of recovery after resection is as follows:

Chin Yuen, a Chinese laundryman, twenty-one years old, entered Dr. F. H. William's service at the Boston City Hospital, October 21, 1898, with a history of intermittent abdominal pain in right upper quadrant for three weeks. There was also an indefinite history of abdominal pain nine months previously, but as the patient talked very little English no definite connection between the two attacks could be obtained. Considerable nausea and vomiting. Bowels regular. Sleep poor on account of pain. During paroxyms he rolled about the bed moaning, and lay preferably on his belly. Pulse 90, regular and of good strength. Heart and lungs negative. A moderately lax,

tympanitic abdomen, tender on deep pressure, more resistant on the right side with increased localized resistance in the right hypochondrium. Two days later there was a leucocytosis of 13,500.

October 27. No vomiting for five days; the face has lost the drawn expression and he no longer cries with pain. Better appetite and sleep.

October 31 (four days later). No abdominal pain since the 27th, bowels regular, general condition much improved and for several days he has been up and dressed ready for discharge, but two days afterward, on November 2, he again had an attack of abdominal spasm and passed some blood at stool. Transferred to Dr. George W. Gay's service under the care of the writer. Examination without ether showed a tumor filling the pelvis and pushing the anterior wall of the rectum backwards, the tumor extending upwards along the right iliac fossa to the right hypochondrium. Spasm of the abdominal muscles. In view of his previous history it seemed best to delay in the hope that he would again quiet down enough to allow operation in the interval, but as the symptoms did not improve operation was performed on November 5th.

Operation. Ether. Incision three to four inches long at the outer edge of the rectus, exposing a sausage-shaped tumor extending from beneath the left lobe of the liver to deep in the pelvis, twelve to fourteen inches in length. Towards the lower end were three gangrenous areas that ruptured on handling, allowing pus and fæces to escape. After clamping the bowel above and below the intussusception, the mesentery was cut parallel to the gut and the vessels controlled with a continuous half-hitch catgut suture, but owing to the abundant vascular supply additional ligatures were required. In order to gain a little room and to save as much of the bowel as possible the gut was disinvaginated for an inch or more at the neck, but after delivery

the circulation was so poor that an additional inch was amputated. After suturing each stump-end with a continuous catgut suture to control the bleeding, an end to end anastomosis was made with two rows of a continuous, right angle silk suture. Owing to the œdema and swelling of the gut at the lower end, a good approximation was difficult, but so far as could be seen the joint was a tight one. The mesentery of the upper portion was bluish, and in one place had a small inflammatory patch, but there was apparently life enough to warrant its being retained. To have amputated more gut in order to exclude this bit of mesentery would have necessitated another parietal incision, which in the patient's condition was not justifiable at the time.

A half pint of salt solution had been injected into the bowel before making the anastomosis; afterwards the abdominal cavity was washed out with hot salt solution, the lower three-quarters of the incision closed with silkworm gut, iodoform wicks and a glass drain being carried in at the upper angle and isolating the region of the joint from the general cavity.

The patient stood the operation, which lasted an hour, fairly well; the pulse at the close being 130 to 140, regular and of fair strength. Strychnia, salt solution and brandy had been given under the skin. Repeated $\frac{1}{4}$ grain doses of calomel and stimulation were ordered.

Good recovery from the operation followed, the bowels moving within twenty-four hours by enema, and broths being retained without vomiting.

Nov. 7. All wicks loosened and some removed under chloroform, the remainder being taken out two days later and replaced by a light packing.

Nov. 11. Intestinal contents, bile and pancreatic fluid escaped from the sinus to-day, but the general condition of the patient remains good. He was then put on rectal feeding, but as symptoms of starvation appeared a second operation was advised.

Nov. 15. Operation. Ether. At the bottom of the sinus a small opening into the bowel lay an inch and a half to the inside of the line of the parietal wound, so a second incision was made close to the median line. The opening into the gut, ½ inch in diameter, lay in the midst of gangrenous and granulation tissue. Considerable time was wasted in trying to close this hole, and then the incision was enlarged and the fresh adhesions broken down in order to allow access to the bowel. Owing to its adhesions and short mesentery it split open, not at the original anastomosis, but apparently in line with the mesentery that was considered doubtful at the first operation. The patient's condition would not warrant prolonged operation, so the ends were sutured together with ordinary silk in mattress sutures passed deeply and at some distance from the edges. Apparently a tight joint was secured. Iodoform gauze drainage. There was considerable shock, but the patient recovered quickly and showed no evidence of peritoneal disturbance. The wicks were removed and he was gaining steadily, when, on the sixth day (Nov. 21), leakage from both wounds appeared. As the general condition continued satisfactory, a third operation was not undertaken until Nov. 26, when, under ether, a fine opening admitting a probe, at the bottom of the median incision, was found lying in firm, healthy tissue. This could be easily and completely closed with fine silk. The entire median wound was then thoroughly curetted, filled with strong permanganate solution and closed with silkworm gut sutures, a small rubber-tissue drain being carried obliquely through the first lateral incision. No shock followed this operation.

Two days later, food, with pancreatic fluid and bile, appeared as of old, but the patient was in good condition, eating solid food and gaining steadily.

Dec. 1. Considerable digestion of the skin, controlled, at Dr. H. W. Cushing's suggestion, by dressing with tinct. benzoin comp.

Dec. 9. General condition improving rapidly; lateral wound closed; apparently no pancreatic fluid, but a little pus from the sinus.

Dec. 17. Again a slight escape of pancreatic fluid and bile.

Jan. 18. Discharge continues about the same, the skin being completely protected by the benzoin.

Jan. 25. Three heavy silk loops (those used at the second anastomosis) discharged to-day. Patient in excellent condition.

Feb. 4. Lateral sinus, which re-opened temporarily, has again closed.

Feb. 18. Patient cheerful and helpful about the ward.

Mar. 17. Discharge is now barely sufficient to moisten litmus paper, upon which it uniformly gives an acid reaction.

Mar. 27. Sinus healed. Patient fat and healthy.

Apr. 11. Wounds firmly closed; patient discharged wearing an abdominal swathe.

At the time of operation, the intussusception which was seen to be in the jejunum was apparently a reversed one, but careful examination later showed that it was a direct invagination. The specimen, when measured after preservation in alcohol, showed that at least thirty-two inches had been excised.

It would have been better to have operated at the time the patient was ready to be discharged, or, at the latest, when the recurrence of the acute symptoms began, as in that way a partial excision might have been substituted for the complete one. No cause in the shape of a tumor or ulcerations could be found.

ARTICLE XI.

# THE GROWTH AND EXTENSION OF CARCINOMA.

By WILLIAM T. COUNCILMAN, M.D.
OF BOSTON.

READ JUNE 13, 1899.

# THE GROWTH AND EXTENSION OF CARCINOMA.*

THE carcinoma is a tumor which arises from epithelial tissues and is characterized by unlimited growth locally and by the formation of tumors of a similar structure in other parts of the body. When we examine its microscopic structure we find that it is composed of two sorts of tissue: 1st, epithelial cells arranged for the most part in solid masses which vary in their size and form, growing in spaces which are called alveoli; 2d, connective tissue which surrounds the alveoli and bears the blood vessels for the nutrition of the cells in the alveoli. This connective tissue is called the stroma. Neither stroma nor blood vessels penetrate the masses of epithelium. When we examine normal epithelial structures we find a very similar arrangement of tissues—epithelial cells growing in the closest juxtaposition with connective tissue, but never penetrated by it. Whether we consider covering epithelium or glandular epithelium, the arrangement of tissue is always the same.

If we take any normal epithelial structure and suppose that the cells at some point begin to multiply with unlimited activity and penetrate into the adjoining connective tissues, we have the structure of the carcinoma. It is not sufficient for the epithelium to proliferate. Epithelial tissues are the most active of any tissues of the body. Both the covering and the glandular epithelial cells are constantly becoming

* These remarks were illustrated by the projection of photo-micrographs.

used up in the course of their physiological activity, and the loss is constantly repaired by the formation of new epithelial cells. This power of regeneration is sufficient not only for the ordinary wear and tear of the tissues, but extensive losses of cells are as easily repaired. Further, the epithelium may be excited to greater activity by various means, and large masses of tissue composed principally of epithelium may be produced. I say principally of epithelium, for, in consequence of a definite law of nutrition, when a tissue is growing actively and requires more food, the old blood vessels will enlarge and new ones be formed. In consequence of this, along with the epithelial masses, considerable connective tissue with blood vessels will be produced. An epithelial growth of this kind repeats the formation of the normal structures and is called typical. There is no penetration of the connective tissue by the growing masses of epithelium. There is no normal prototype for the structure of the carcinoma. We express this by saying that the simple hyperplasia we have been speaking of is typical, while the carcinoma is atypical.

When we compare the epithelium in the different epithelial structures of the body, we find differences in the size and form of the epithelial cells, and in their arrangement. These differences are to a certain extent apparent in the carcinomas which arise in different parts, and from the histological examination of the tumor we can say with considerable accuracy from what tissue it has arisen. To explain the growth of the carcinoma it will be necessary to consider for a moment the structure of the connective tissue. This is essentially formed of fibres, between which are cells. It carries the blood vessels everywhere, and in it the lymphatics arise. In the tissue the cells lie in spaces, which communicate with adjoining spaces, and from the enlargement and coalescence of these spaces the lymphatics are formed. If we suppose that a carcinoma begins its forma-

tion by the epithelium growing outward into this tissue, then the growing cells of the tumor at once find their way into this system of communicating spaces and continue to grow in them. Every mass of epithelial cells is within such a lymph space of the tissue.

When we examine sections from the centre and periphery of a carcinoma we see certain differences in structures. In the periphery the cells stain more sharply and more of them show the peculiar changes in the nuclei, by which we recog nize cell division, than do the cells in the centre. The same thing is seen more clearly when we make a section directly through a small carcinoma. The most active growth is in the periphery. In the centre the alveoli are small, the cells have degenerated, and in some cases have disappeared altogether. All the blood vessels of a tumor enter from the periphery, and here the best nutrition is offered the cells. The atrophy which goes on in the centre is partly to be accounted for by the compression and occlusion of the vessels by the growing epithelial masses between which they must pass, and partly by the peripherial masses making more active demands for the nutriment which the vessels contain. The size of the single alveolus also has limits. The epithelial masses receive their nutrition also from the periphery because blood vessels do not penetrate within them. In consequence, the cells on the outside proliferate and the newly-formed cells are pushed into the interior, and with the enlargement of the mass are further and further removed from the source of nutrition. As the alveolus enlarges, degeneration takes place in the central cells. Each alveolus then becomes in its growth a type of the whole tumor. The malnutrition of the centre of the tumor affects not only the epithelial alveoli but the connective tissue stroma. It loses its characteristic structure, the fibres blend together into a dense mass, and the cells atrophy. It contracts just as cicatricial tissue does, and hence arises the

central depression so characteristic of carcinoma in certain places.

Certain names are given the tumor to express the differences of relation between stroma and alveoli. The hard dense tumor due to central degeneration, having small alveoli and dense stroma, is called scirrhus. The softer tumor, with larger alveoli and softer stroma, is called medullary. Both scirrhus and medullary are seen in the same tumor. The hardness of a tumor is frequently only an evidence of the activity of the peripherial growth.

Every structure gives way before the growing epithelial masses. This is partly due to pressure and partly to the fact that the epithelial cells demand the nutrition which is provided for the opposing structures. Even the non-maligant tumor presents higher claims for nutrition than the normal tissues. It is not uncommon to find a fatty tumor in an individual so emaciated that almost all the normal fat has disappeared, but the fat cells of the tumor not only suffer no diminution in size and number, but continue to increase. The carcinoma not only grows peripherially as a solid mass, but in various places small masses of cells push out independently into the tissue and form new centres of growth which gradually coalesce with the central mass. The contraction of the tumor due to central degeneration is particularly seen in carcinomas of the alimentary canal. In this the central part of the tumor is connected with the lumen. The degenerated necrotic portions are invaded by bacteria, which find there a suitable soil for growth, and inflammation and ulceration results. In the stomach the necrotic portions are digested by the gastric juice. The stricture of the intestine results both from the masses of the tumor pushing into the lumen and from the contraction which constantly accompanies the degeneration.

The carcinoma not only tends to grow locally in the way we have seen, but similar tumors are formed elsewhere. In

carcinomas these secondary or metastatic tumors are found in those lymphatic glands which are connected with the primary tumor by lymphatic vessels. In carcinomas of the alimentary canal, the secondary growths are found not only in the lymphatic glands but in the liver, the connection being by blood vessels of the portal system. The metastases repeat the structure of the primary tumor. Differences in the size and arrangement of the cells which characterize the primary tumor are found in the secondary. The same tendency to degeneration and the same form is seen in the secondary. Something capable of producing the same growth must be carried from the primary tumor to form metastases. We can trace the growth of the metastases much better than we can the growth of the primary tumor. By examining those lymphatic glands of the axilla removed with a carcinoma of the breast, which to the naked eye present no enlargement or deviation from the normal, the beginning of the metastasis can be seen. It begins in the periphery of the gland where the lymphatics coming from the primary growth enter. Cells of the same character as those in the primary growth are found in the lymph sinuses, and they grow here, the remainder of the gland forming the stroma.

The growth advances along the lymphatics in two ways. Solid masses of cells probably connected with the primary growth may push their way along the lymphatic vessels. I have seen all the lymphatic vessels of the scrotum injected with carcinoma in this way, separate nodules being formed at intervals by more active proliferation of the cells in the lymphatics. In the carcinoma of the stomach it is not at all uncommon to find the lymphatics of the peritoneum over the tumor filled in this way with masses of cells. Secondly, not only can we trace these masses of cells in the lymphatic vessels, but by examining the tissue which is in the neighborhood of a carcinoma and which is apparently not

affected, we can often find single epithelial cells within the lymphatics. In certain cases the tumor cells may push their way into blood vessels. In carcinoma of the alimentary canal metastases both by the lymphatics and blood vessels is the rule. This is probably to be explained by the presence of such numerous and thin-walled veins in the tissue.

We may certainly regard the metastases as due to the conveyance, by either the blood vessels or lymphatics, of cells from the primary tumor to a distance. The proof of this is the close connection of the cells of the tumor with the lymphatic system, the presence of cells showing similar structure mode of growth and tendency to degeneration in the secondary tumors, the beginning of secondary tumors in the lymphatic glands where the lymphatics enter the gland, and the presence not only of cell masses but of individual cells in the lymphatic vessels. The full recognition by surgeons of the intimate relation between the cells of the tumor and the lymphatics has led to those methods of operation which have so reduced the danger of recurrence. We know that no carcinoma can be found so recent in its development that the lymphatic glands belonging to the part may not be affected. Hence these are removed with the primary tumor. Further, that the way of extension is by the passage of cells from the primary tumor or along the lymph vessels. It is of no use to remove the primary and secondary growth and leave a long street between the two which almost certainly contains straggling cells capable, each of them, of producing a new growth. Hence tissue containing lymphatic vessels connecting primary growth with lymphatic glands is removed as far as possible. Cells from the tumor, each capable of developing a new tumor, may be implanted in the tissues by the knife in the course of operation; hence the care exercised in avoiding incision into the tumor in the course of operation.

When we come to the question of the essential cause of carcinoma we enter the domain of theory. Of these theories we have, first, the influence of trauma, which is supposed to irritate tissues and to cause them to proliferate. Trauma alone has little to be said for it. The infrequency of carcinoma in those parts which are most exposed to trauma and its frequency in those parts least exposed speaks against this. No parts of the body are so exposed to trauma as the hands and feet, no parts less exposed than the mammary gland and the uterus. Even in the pessary days of gynecology carcinoma of the uterus was not made more frequent. Carcinoma of the lower lip, due to smoking, is a favorite example for the traumatist. Why the infrequency of carcinoma in the upper lip, which must be just as much exposed? It would take an accomplished juggler to hold a cigar in the lower lip. The appearance of the tumor at the period of life when traumas of all sorts are less apt to take place, speaks also against it.

2d. The theory of Thiersch that the essential element in the production of a carcinoma is a degeneration of the connective tissues, in consequence of which the epithelium more easily grows into it. Such degeneration, if true, should be capable of demonstration, but has not been demonstrated.

3d. The theory of embryonic residues of tissue included in the normal tissues and capable of the enormous capacity of growth which characterizes embryonic tissue. This theory, though not original with Cohnheim, was taken up by him and advocated with all the force and charm which characterizes his work. In favor of this it must be said that there is much analogy between tumors and embryonic tissue, and that such remains of embryonic tissue may exist in the body and that tumors may develop from them. Considered purely as a theory, there is undoubtedly more to be said in favor of it then of any of the others. A modification of this theory has been advocated by Ribbert, who

supposes that not only such embryonic tissues develop into tumors, but that when masses of epithelium become separated from their organic connection with other cells they may develop into carcinoma. There is danger in even discussing these theories that one may be tempted to advance one.

Probably the theory which of late is having the most attention paid it is that of the parasitic origin of carcinoma. From a purely theoretical point of view there is much which speaks for and more against it. It must be considered a theory, for as yet parasites have never been demonstrated in a carcinoma, or certainly not in such a connection with it that they should be considered as causal. From a theoretical point of view, there are certain things which at first sight seem to favor the parasitic theory. First, the apparent increase in the number of deaths caused by the malignant tumors. If these tumors are due to embryonic causes or trauma, or such constantly operable causes, there would be no likelihood of increase. If due to parasites they could, like the other infectious diseases, show periods of increase and decline. There can be no doubt, from the numerous articles which have appeared on this subject, that the general mortality statistics show on the face a marked increase in cancer mortality in the last twenty-five years. It is possible to take mortality statistics and prove many things by them. Only those who know how mortality statistics are gathered, who see the crude returns before they are interpreted by the clerks in health offices and arranged in fair columns of figures, know their full unreliability. The exponents of protection and free trade have shown what can be done with the fairly accurate economic statistics. The more accurate statistics of insurance companies do not show this increase. The apparent increase deduced from the general statistics can be referred to a number of causes. There has been an increase in longevity, due principally to diminished mortality in the infectious diseases. Carcinoma is essen-

tially a disease of later life, and more people reach what may be termed the carcinoma age. The diagnosis of carcinoma, especially of the internal organs, has become more accurate, and deaths are now properly attributed to this disease which were formerly put down under other heads. Deaths from carcinoma were formerly concealed as much as possible, so great was the horror of the disease and the fear of heredity. Hospital surgeons gain a general idea of increase from the number of cases who come to the hospital for operation, due to the great success which now attends such operations. The disease carcinoma in some respects shows a similarity to the chronic infectious diseases. Like these, it commences in some locality, and from this it extends to other parts by means of the blood or by lymphatic vessels. In carcinoma, as in infectious diseases, there develops after a variable time, a condition of wasting and malnutrition which is termed cachexia. When we examine more closely, the analogy is not so evident. In the first place, the tissue lesions are totally different. There is a great deal of similarity in the lesions of all the infectious diseases. They consist in a combination of degeneration and proliferation of tissue. In certain of them, as in tuberculosis, the character of the degeneration produces a lesion typical from which it is possible to make a diagnosis, though not with absolute certainty. In all, the new tissue formed is of an indifferent type and of the general character of the granulation tissue of inflammation. In the metastases the same general character of lesion is produced as in the primary. Opposed to this, in carcinoma a definite tissue is produced consisting of epithelium growing into a connective tissue stroma. It is not an indifferent tissue which might arise from proliferation of the connective tissue, but is a definite tissue formation consisting of epithelial tissue in a definite relation to connective tissue. Moreover, the metastases show the same type. It is possible to have in

the liver secondary to an adeno-carcinoma of the rectum an almost perfect reproduction of the glands of Lieberkühn. If the cells are small in the primary growth, if they have any peculiarity in arrangement, if they show any peculiar form of degeneratiou, the same is true of the metastases. The metastasis which has been used by the exponents of the parasitic theory as showing the analogy between carcinoma and infectious diseases cannot be produced by parasites alone. In the infectious diseases the primary lesion is usually at some point which is open to infection from without, as the lungs in tuberculosis. In carcinoma the disease is most frequent in those parts in which the ordinary infections are most rare. Certain peculiar bodies are found included in the cells of carcinoma which have been considered as parasites. It is perfectly true that we do find in the epithelial cells of carcinoma small structures of whose nature we are for the most part ignorant. We do not better the situation by saying, therefore, they must be parasites. They show no definite size or form, they have no definite reaction to the analine dyes, and if parasites, they differ from any known type. It is not impossible that carcinoma may be due to the invasion of the tissue by a parasite. If true, the invasion must take place only in the latter half of life, in places least exposed to invasion of the ordinary infectious agents, the parasite must enter every cell causing it to proliferate and grow in a peculiar way, and must be carried with the cell in other parts of the body and there again cause the same peculiar cell growth.

The enormous advance in our knowledge of disease in the last twenty-five years has scarcely affected the malignant tumors. The classification has been improved and we know their structure rather better, and that is about all. There is need now, not of any new theory, but of close investigation. The parasitic theory must be taken up and definitely proven or set aside. The question of their inoculability

must be determined. The investigation should cover not only the malignant tumors in man, but possibly a more fruitful field may be found in the tumors of lower animals, particularly the common tumors of fish. Such investigations are being carried out in various places in other countries and in this, and we confidently believe that the results of these investigations will lead to an increase of our knowledge and with this, increase in our power to relieve.

ARTICLE XII.

THE ANNUAL DISCOURSE.

# REALISM IN MEDICINE.

By ARTHUR T. CABOT, M.D.
OF BOSTON.

DELIVERED JUNE 13, 1900.

NOTE.—At an Adjourned Meeting of The Massachusetts Medical Society, held Oct. 3, 1860, it was

*Resolved*, "That The Massachusetts Medical Society hereby declares that it does not consider itself as having endorsed or censured the opinions in former published Annual Discourses, nor will it hold itself responsible for any opinions or sentiments advanced in any future similar discourses."

*Resolved*, "That the Committee on Publications be directed to print a statement to that effect at the commencement of each Annual Discourse which may hereafter be published."

# REALISM IN MEDICINE.

Mr. President and Fellows
of The Massachusetts Medical Society:

When I received the compliment of an invitation to address you to-day I first thought that here was an opportunity to present some surgical subject which would have a general interest. Before I had settled on a theme, however, I remembered that this was the year 1900, and I felt that it would be plainly unfitting, at our last meeting in the nineteenth century, to ask you to confine your thoughts to any narrow field of research. For we are just finishing the hundred years in which medicine and medical study have done more for the world than had been accomplished by the disciples of Æsculapius in all previous time.

Wallace, in his recent book entitled "The Wonderful Century," draws a striking comparison between the achievements of human endeavor in the nineteenth century and in all preceding ages. He takes account of the discoveries which have been of sufficient weight to turn the current of thought or to profoundly modify the lives of men. In parallel columns he places these lists of notable

events; on one side those which preceded the year 1800, and on the other those which followed it. Certainly he is justified in calling this a wonderful century, for twenty-four important discoveries are credited to it, while he has found but fifteen discoveries of similar magnitude in all past time.

However we may differ with Wallace in some of his estimates, we must agree, I think, that this comparison is substantially a fair one, and must recognize that, measured by such tests as we can apply, the world and the conditions of life upon it have been more altered by man in the past century then in all time that had gone before.

In no realm of human thought has this recent advance been more rapid than in that of medicine, and in none other has it been of such wide and lasting benefit to humanity.

When we name surgical anesthesia, cellular physiology and pathology, antiseptic surgery and the germ theory of disease, we have an array of gifts for which the human race may well feel grateful to the medical men of the nineteenth century. Looking back through all past time, we find that in the seventeenth century Harvey discovered the circulation of the blood, and that in the eighteenth century Jenner introduced vaccination. Here the list of medical contributions of the first rank ends; four in the present, two in all past centuries. And of these two, vaccination, while working great good, did not admit of scientific explanation or understanding until the investigations arising out of the germ theory

opened our eyes to the significance of acquired immunity.

It would be manifestly impossible, in the time at our disposal, to review in any detail the progress of medical science in the three generations just past, and I do not mean to task your patience with an attempt at such a recital. But I think it may be well worth our while to take note of the discoveries which mark epochs in medical progress and which have occurred within the memory of many of those here present. A consideration of the direction of the research or study which led to them may help us to draw from them lessons, or deduce from them curves of thought, which will guide us along the best lines in the century that we are just entering.

A brief consideration of the history of medicine shows that it may, for purposes of study, be divided into two periods. Of these, the first is that long period from the birth of known time up to the beginning of this century, during which Idealism prevailed and theory succeeded theory only to be in its turn superseded by some newer invention of a more ingenious thinker. The second period is that of Realism, in which theories are not allowed to stand unless they can be shown to rest on demonstrable facts. It is not claimed that this, the inductive method, had not been used in medicine prior to the present century. Far from it, for the two already mentioned achievements of the past, namely, the discovery of the circulation of the blood and vaccination, were both obtained by men

following realistic methods, searching for facts and basing their theories on these facts only after they had been thoroughly tested.

It can be fairly asserted, however, that it is only within the past hundred years that inductive reasoning has become the prevailing influence in medicine, and that abstract ideas have come to be discredited. Now and then, even in the remotest times, men who were born observers attained some insight into the realities of things. Such men often solved the practical difficulties that were presented to them in the best manner and introduced rational methods of treatment which did much to keep medicine out of disrepute during that long period when error masqueraded in the cloak of philosophy. The questions that were intelligently handled in those early times were mostly anatomical or surgical and dealt with the visible or easily accessible parts of the body. The moment that obscure functions or the hidden causes of disease came under discussion, then the all-pervading idealism resumed its sway and the wildest speculation and theorizing prevailed.

Thus at the time when surgeons met the serious difficulties of an operation for stone in the bladder in a rational and enlightened manner, their medical brethren were treating pneumonia, typhoid fever and other diseases that taxed the endurance of their patients to the utmost, by bleeding and other methods of depletion, which still further reduced the strength so greatly needed.

It is true then of all time that medicine has owed its successes to its use of inductive methods. It has

reached lasting results only when it has rested its theories on accurately observed facts. We recognize the human senses as being on the whole, reliable observers, worthy of acceptance as witnesses to the facts.

No one would for a moment deny that our senses are fallible and often deceived; but they are the only witnesses we have, and we must make the best of them. They must not be easily trusted, and their observations must be constantly verified by independent investigations. This constant re-examination and verification or disproval of work done has been a marked feature of medical progress in this century.

Louis, the great clinician, who was active at the time when idealism was losing its hold and realism was coming to the front, said, "As often as I have formed an *a priori* idea and had afterward opportunity to prove the facts, I have invariably found that my idea was false." Here is the testimony of an honest man, striving after the truth and forced to recognize the fallibility of the human mind when acting without the constant control of the observant senses.

The present consistent and ever advancing progress of medicine toward a solid and enduring position among the arts and sciences of the world dates from the time when medical men came to Louis's way of thinking, and ceasing to be content with the so-called systems of medicine which had their origin in the minds of ingenious closet naturalists, sought to establish a foundation of fact for the superstructure of their thought.

It was then that they ceased to be animists, vitalists, Brunonians or animal magnetists, and began to be scientific investigators, searching after the truth with the best powers of observation that nature had put at their disposal.

They were no longer content with the pleasing and fleeting shows of the imagination, but when they found a fact which kept its integrity through all the tests that their ingenuity could apply, they duly prized it and put it by, to use when they could make it applicable. Such tested facts and the beliefs founded on them were kept under constant scrutiny, and if an improved method of investigation was discovered, the previously trusted facts were re-examined and rejected if found wanting.

The scientific spirit is one of skepticism, hard to convince and very annoying to the intuitive thinker. But the nineteenth century has shown us that this patient piling of fact upon fact builds up structures of an interest and beauty before which the wildest dream of the idealist pales, and with a quality of durability which is in strong contrast to the ever changing beliefs of the past.

The history of surgery affords, perhaps, as good an illustration as is possible of the triumph of the materialistic, the modern scientific method over the old idealism.

Who can tell us better the aspirations and hopes which seemed justified under the old régime than Ambroise Paré? No one better represents the high ideals of the surgical art in the past than he.

In 1575 he said: "God is my witness, and men are not ignorant of it, that I have labored more than forty years to throw light on the art of surgery and bring it to perfection. And in this labor I have striven so hard to attain my end, that the ancients have nought wherein to excel us, save the discovery of first principles: and posterity will not be able to surpass us (be it said without malice or offence), save by some additions such as are easily made to things already discovered."

To-day this prophecy sounds ludicrously short-sighted; yet it remained true for almost three centuries and forecast with considerable accuracy the course of surgery up to the time of the scientific upheaval which realism brought about. Could Paré come back to earth at this time, what joy he would take in the triumphs of modern surgery, for he loved his art truly and disinterestedly, and he would freely forgive the manner in which his prophecy has been belied. Not the least part of his pleasure would be in finding that with all the extraordinary advances that surgeons have made, they still hold fast to the ligatures that he gave them for the control of hemorrhage. Paré was the great realist of that day. He delighted in close observation and dared to follow what he saw.

To trace a little in detail, now, the paths along which the modern advances have been made, we find that at the beginning of the century the human senses, while recognized as credible witnesses and depended upon in a measure for the investigation of truth, were found to lack the acuteness neces-

sary for penetrating many of the secrets of Nature; and human ingenuity began to be turned to efforts to assist the preceptions and to widen their scope.

The power of the eye had for a long time been increased by the use of lenses, and the wish to aid its penetration into the depths of the body led to the invention of the ophthalmoscope, the laryngoscope, the cystoscope and other combinations of mirrors and lenses which brought the various cavities of the body within the reach of direct observation. Efforts in this direction will not pause until man ceases to be an inquisitive animal. Every year sees improvement in all such methods of investigation and a consequent widening of our knowledge of the hollow organs. Finally, at the end of the century, the remarkable discovery of Röntgen has enabled the vision to penetrate and discover the secrets hid in solid tissues.

In the meantime, the sense of hearing was not neglected. Something was known of percussion in the seventeenth century, and a rude auscultation of the borborygmi in the bowels had been practised before that; but the thorough and efficient use of these methods of diagnosis waited for Laennec, who, in the first half of this century, by his invention of the stethoscope, greatly aided the sense of hearing and opened the way for the modern practice of auscultation and percussion which has reached such perfection that the relative positions of deeply lying organs can be accurately made out and the lightest whisper in lung or artery is heard and understood.

Even the sense of touch has had its range of usefulness widened. For modern surgery affords so many opportunities for the correction of ideas gained through palpation, that the accuracy of deductions drawn from what it is possible to feel has been greatly increased, and a modern abdominal or pelvic examination is as great an advance on the less assured methods of a century ago, as are the modern means of locomotion over the old stage coach.

Extraordinary as have been these advances in the investigation of living conditions under the earnest and constant pressure of clinical workers, it is when we turn to regard what has been accomplished by the post-mortem study of tissues healthy and diseased, that we approach that part of achievement in medicine which will make this century memorable.

Much had been done in the way of accurate observation of morbid conditions prior to the present century, but it was after the improved lenses of the modern microscope made possible the close study of the individual cells, that medicine made the forward stride which placed it in the ranks of exact science. Many men contributed to this result; for with the awakening of interest in the objective study of medicine, the laboratories, where such existed, were thronged with eager investigators, and if the time served, many of these might be mentioned with honor and reverence, for they advanced nearly abreast into the newly opened regions of cellular pathology.

It is impossible, however, to speak of this movement without mentioning the name of Rudolph Virchow, who will ever be remembered as the one who stepped forward from the ranks and led this advance. I well remember my first impression of Virchow when I went to Berlin in 1876 to study in his laboratory. He was not at first sight an imposing figure, and one would not have picked him from a crowd as the foremost medical thinker of the time. Any sense of disappointment on this score was, however, soon dispelled when he was seen at work. If I had brought no other impression from my foreign study I should have felt amply rewarded by the opportunity of seeing that trained intellect, backed by unrivalled powers of observation, leading the way through the most complicated pathological problems and making that way so clear that we wondered we had not seen it for ourselves. His eye seemed to penetrate to the centre of the organ he was examining, and we students used to say it was as good as a No. 7 Hartnack objective. One might well feel that to have been associated with him was a liberal education in medicine.

The habit of verifying, with the microscope, appearances dimly seen with the naked eye, and studying out the cellular changes involved, laid the foundations of modern pathology. At the same time, the optical instrument makers were constantly improving the lenses and methods of illumination in the microscope, so that it was possible from year to year to get a more and more

accurate idea of the cells and of the changes that occurred in them as the result of morbid processes. Embryology too, threw much needed light on the life history of the cell and upon the characteristics of different tissues.

This patient work of investigation required for its proper prosecution institutions devoted to the scientific study of medical problems, and it also required adequate means for the support of the investigators. Such institutions, and the necessary endowments, hardly existed in this country until the last quarter of this century, so that the earlier American students, who wished to be properly instructed in these higher methods of research, were forced to go abroad to seek the opportunity. Especially in Germany they found good laboratories and inspiring teachers, and they came back after their probation, inoculated with the skeptical, investigating Teuton habit. Even if they were soon plunged in the every day work of their profession and found but little time for the further prosecution of purely scientific work, still this habit of inquiry and of constant scrutiny of conditions passing under their eyes clung to them and was of the greatest service in their practice and in such study of their cases as they could make.

A man with his standard of work thus raised was no longer content with a name as the explanation of a set of symptoms, but felt driven to seek out and understand the morbid process which caused the observed phenomena and, if possible, to learn the cause of that morbid process. He had

thus ceased to be an empiric and had become a scientific practitioner of his art.

The arousing of this spirit throughout the medical world has been perhaps the best gift of this century to medicine. The establishment of the cellular histology and pathology marks one goal reached by this movement which is pressing on with a still accelerating speed.

We now come to speak of the second great contribution which this scientific spirit has made to the century. I refer to the germ theory of disease. The manner in which this discovery was made and in which the facts thus far established have been reached, is most encouraging, and awakens inspiring hope for the future. For it was not revealed by any flash of inspiration to a closet thinker, nor was it accidentally stumbled upon by an erratic investigator groping far ahead of his time. On the contrary, it came as the logical result of concerted human effort, stepping forward constantly from fact to fact. It was the most complete possible demonstration of the worth of this sort of study. There could be no more convincing proof of the value of laboratory work than this. When the cause of a disease is discovered by such investigation and its authenticity is shown by its power to reproduce the same disease, we at once feel that we have here solid ground from which to start for further researches.

It was this achievement, perhaps, more than any other, that informed the world that at last medicine was ceasing to be merely an art and was taking its place among the exact sciences.

We cannot speak of the germ theory without thinking of certain illustrious men. The names of Pasteur and of Lister will at once occur to you. We must not forget, however, that long before Pasteur's discovery of the influence of micro-organisms in producing fermentations, important work had been done by Vogel, Davaine, Schulze, Chevreuil, and many others. It remained for Pasteur to so illuminate the facts that thenceforward no one could doubt that the minute organisms associated with putrefaction and other fermentative changes were really the cause of those changes and not an accidental accompaniment or result of them.

Lister was deeply impressed by these discoveries. It had long been observed that subcutaneous injuries were quick to heal and followed a usually afebrile course, in marked contrast to the suppurations and fevers that accompanied the healing of open wounds. He was at once struck by the important bearing that Pasteur's discovery had upon this hitherto unexplained fact, and conceived the idea that the putrefaction produced in animal fluids by the action of organisms, might be responsible for the unfavorable course of healing in wounds exposed to the air. To test the correctness of this hypothesis he devised means of excluding organisms from wounds. The success of his efforts in this direction compelled the attention of surgeons and led by rapid steps through antiseptic to the aseptic surgery of the present day.

The discovery of this application of the germ theory threw the door wide open for the surgeon

to enter safely into the inner recesses of the body and to interest himself in the functions and disturbances of organs that had before been as sealed books to him.

The triumphs of the new surgery might well fill a discourse. It is with difficulty that I turn away from so fascinating a subject. To one whose surgical activity began with the introduction of antiseptic principles and who has grown up with the growth of modern surgery, it might well be forgiven if he dilated somewhat upon the triumphs of this golden age in which the power wielded by the surgeon has so wonderfully grown year by year. We are constantly urged forward by the encouragement, Be bold; Be bold, and but rarely are checked by the admonition, Be not too bold.

I cannot, however, allow myself the pleasure of such a digression, but must be content to point out that all this splendid achievement is one step in the onward progress of medical science, and that we owe it to the patient, plodding work of investigation that had gone before.

The interest of every laboratory, and indeed almost every microscope, was now turned to the scrutiny of fluids and tissues, for the purpose of discovering the individual organisms which caused each disturbance. One after another the bacilli, streptococci and staphylococci were ferreted out and classified, and with the aid of solid gelatine media they were presently obtained in pure cultures and their individual characteristics were made demonstrable.

It was now found that special organisms produced special diseases with such certainty that in cases of doubt the detection of the microbe involved in a morbid process at once settled the character of that process. Thus a malignant pustule could be at once distinguished from an ordinary furuncle or carbuncle and the appropriate treatment could be adopted early.

In the presence of such exact knowledge the nomenclature of disease improved, and instead of speaking of erysipelas, lymphangitis or cellulitis it now became usual to say a streptococcus infection of such and such an extent. We ceased to use the terms scrofula or consumption, but began to speak of tuberculosis of this or that organ, and to demand a demonstration of the correctness of our diagnosis by the recognition of the bacilli.

These well-known instances of our changed conceptions of familiar diseases are selected as illustrations of the fundamental alteration of our habits of thought wrought by these newly learned facts.

The next natural step in the investigation of the action of micro-organisms within the body was the study of the effects of these organisms on the tissues and of the provisions that nature made to resist their deleterious action and to limit their growth. Considerable progress has been made in this research.

It has been shown that the blood serum has a certain amount of destructive power and that the white blood cells are also active in the attack upon them. It has also been found, in the case of cer-

tain germs, that their growth in the body produces a future immunity of greater or less duration, and in the case of the Klebs-Lœffler bacillus this immunity has been traced to the blood serum, which acquires, under the influence of the organisms, a positively antidotal power.

The action of the antitoxic serum obtained from horses treated with the diphtheritic poison is so well known to you all that it would be both superfluous and tedious to dwell upon it here. It is, however, perhaps the high water mark reached by this modern investigation of disease. To learn the ultimate cause of a disease, and then, by a study of this cause, to obtain the knowledge which enables us to meet and vanquish the morbid elements after they they have obtained a foothold in the body, is certainly an achievement to be proud of.

It is natural that other organisms should have been studied with the object of discovering similar antitoxins. Efforts in this direction with the tetanus bacillus have yielded results that are decidedly hopeful, and the organisms of relapsing fever, cholera, the plague and tuberculosis have also yielded sera which seem to have some power in either checking or protecting against the parent disease. These are results most encouraging in view of the comparatively short time during which investigations in this direction have been under way. Much, however, remains to be done.

I now wish to turn aside for a moment to speak of an achievement which has been of incalculable benefit to medicine and mankind, but which lay

out of the line of the regular advance of scientific medicine and stands by itself as a gift made by empiricism. I allude to the discovery of surgical anæsthesia. No consistent, experimental study preceded and led gradually up to the final triumph. To be sure, an unconsciousness which, if not too deep, could be recovered from, had been produced by carbonic dioxide, and as early as the year 1800 it became known that nitrous oxide gas would produce a temporary unconsciousness to pain, but even this latter agent, which has since been shown to have some degree of efficiency, was at that time deemed so unsatisfactory that it attracted little attention.

At this time, when the world was unprepared for such good fortune, the knowledge of the anæsthetic properties of sulphuric ether was given to Thomas G. Morton, a Boston dentist, who was without scientific training, but who was possessed of the idea that surgical anæsthesia was possible, and who in his effort to compass it, was ready to take any hint that came to his ears as to an agent by which it might be obtained. His good luck led him to hear that sulphuric ether, when inhaled, produced a temporary unconsciousness. Knowing no more than this, ignorant of the possible dangers of the drug, he risked his reputation and future peace of mind by trying it. Had ether possessed dangerous qualities and caused the death of the first patient, or had that patient been so unfortunate as to die from asphyxia, a mishap that might well have happened with the then in-

complete knowledge of proper methods of etherization, Morton would probably have gone to gaol and later to a dishonored grave. He took those risks and Fortune smiled on the brave, albeit reckless, man. His was the venture and his is the honor.

Surgical anæsthesia was thus discovered, as it were, by accident. It came as a revelation rather than as a result of consecutive steps leading to that goal.

It is interesting to notice, too, that it was revealed at the very time when experimentation upon animals was beginning to be necessary to medical research. It seems to have been pre-arranged that when man's development reached the point of needing to study the phenomena of life upon the living, then his intelligence should compass the relief of pain.

This discovery, as I have said, lay to one side of the line along which scientific medicine was advancing, but it has been of incalculable aid to that advance. How greatly the triumphs of aseptic surgery would have been curtailed if surgical anæsthesia had not been attained. And in experimental medicine it has done far more to alleviate the pain of necessary operations. Without it, many of the most important investigations would have been impossible, for the delicate dissections and manipulations necessary in physiological research could not have been made on animals conscious and struggling.

I have presented to you this brief and incomplete sketch of the great movement of medicine under the guidance of Realism, to show how it has steadily proceeded along a path without a break, or rather the progress has been more like that of an army penetrating an enemy's country, where different columns, mutually supporting each other, advance along parallel lines, meeting success, now here, now there.

Cellular pathology put us in a position to appreciate and understand the action of germs, and the work in bacteriology opened ways in which the student of the cell could make still further conquests.

I have not tried to do more than point out the grand strategy of the forward movement and the important places that have been taken. Many of the lesser achievements in this advance have however been essential to the success of the greater.

Physiology, physiological chemistry, pathology, bacteriology, embryology, anatomy, clinical medicine and surgery, have all, within their own fields of action, kept abreast of the general movement. Men trained in Science, skilled investigators, have occupied the firing line, constantly pushing forward, and as fast as they have gained new territory the practitioners of medicine have moved in like settlers and made it their own.

After this hasty review of the general course of medicine in this century, it should be of especial interest to us to study the part which America has played in promoting and in keeping step with the advances that have been made.

At the beginning of the century the main idea that an American student had in entering upon the study of medicine was to learn what he could of the care and treatment of the sick, with so much understanding of anatomy, physiology and pathology as could be made of service in his pratical work. The teaching in the scientific branches of medicine was, as I have said, very meagre in this country, so that a young man, wishing to be thoroughly abreast of the times, was forced to seek instruction in the laboratories of Europe. The result of this was that a well-trained American physician had usually passed two years abroad acquainting himself with the methods of the best teachers of the world.

The lack of opportunity at home, forcing men to travel, had its partial recompense. For the young American, travelling from teacher to teacher, going for each part of his education to the place where that was best taught, learned to look at things in a broader way than would have been the case had he found all that he required in one school.

This increased breadth of view was at once reflected in the medical practice of the men so trained, and long before American physicians began to take their proper place as scientific investigators they developed in their ranks some of the best practitioners in the world.

Many men came back from their foreign travel filled with a desire for research; but the absence of facilities and the need for self support stunted their

aspirations in such directions. It was not until the last quarter of the century that the use of labaratories became so manifest that they began to spring up and to receive adequate support in this country. I cannot, even in so condensed a review of this movement, forbear to speak of the pioneer work done at the Johns Hopkins Hospital under the inspiring lead of our guest, Prof. William H. Welch. The wise provisions of that foundation gave the Johns Hopkins Medical Faculty the almost unique opportunity of putting their school on the right line from the start. And thanks to the wisdom and energy of Professor Welch and his co-workers, the scientific investigation of disease at once took its proper place in that institution. Since that time the constant help that laboratory investigation gives to the clinical study of disease has appealed so strongly to the practical American mind that pathological laboratories have sprung up in every direction and are still constantly on the increase. These have been of the greatest value in raising the scientific quality of the work in many of our communities. Such laboratories have given men inclined to research opportunity for pursuing their studies, and have also enabled many clinical workers to acquire a knowledge and facility in scientific methods. This training has been of great service in their practice. They have gained a clearer understanding of morbid processes and have come to have a personal acquaintance with the germs against which they are constantly waging war. When a man thus

trained speaks of a bacillus he has in his mind a distinct picture of its appearance and its characteristics. He knows where to look for it and what effects to expect. If it is a pneumococcus he watches the serous membranes and early detects its migration to them. His horizon has become wider and his vision more acute.

With the appearance of laboratories the need of endowments for pathological and other scientific work has been brought home to our people. Practical business men see that the researches in the higher branches of medicine bear tangible and most valuable results; that by them the health of the community is improved and protected. They are thus encouraged to invest in a movement which has such evident good effects in the present and that holds forth still brighter promises for the future. Considerable sums of money have been drawn into these enterprises and have already done much good. They have, however, been but as a drop in the bucket which we wish to see filled. This work, by its interest, attracts the best minds and the world should discharge its proper obligation to them and should see that they are adequately cared for.

The administration of laboratories and the prosecution of complicated investigations is expensive.

At present I see no direction in which charity to the human race can better expend itself, with full assurance of lasting good, than in assisting the prosecution of such studies whose benefits are shared by all peoples and go down to remotest posterity.

The perfecting of a system under which the scientific investigation of disease may flourish should be the constant aim of all American physicians. The profession of this Commonwealth have already taken steps in this direction, of which mention may be here appropriate.

A quarter of a century ago we had at the Harvard Medical School, a chemical laboratory, a physiological laboratory, a dissecting room and a small pathological laboratory. Students had fairly good opportunities for the study of chemistry and anatomy, but they had no regular laboratory instruction in either physiology or pathology. Both of these important branches were taught by lectures to large classes in which each individual had but little personal contact with the professor, or with the phenomena described. The pathological instruction up to that time and till a considerably later period, was given by men who depended on clinical practice for their support and gave what time they could spare from this to their own scientific studies and to the instruction of the students.

It would take too long to describe in any detail the gradual changes by which this condition of things grew and expanded into that which exists to-day; but a statement of the present provisions for research and teaching in the Harvard School will give an idea of the rapidity with which this movement has advanced.

The physiological laboratory, with a professor, an associate professor and a greatly increased

corps of teachers, is now thrown open to the students who receive personal instruction in small classes. There is a laboratory for embryology, and histology, where the development and the finer anatomy of the tissues is taught to small classes, by the professor, the assistant professor and other assistants.

There is a good-sized pathological laboratory under the direction of a professor who gives his whole time to study and teaching, and he has in addition to this the control of the clinical laboratory at the City Hospital, which is under the supervision of the assistant professor.

The clinical laboratory at the Massachusetts Hospital is also available for teaching purposes, and being under the direction of one of the instructors in pathology, makes very substantial contribution to the opportunities that the students have in this branch.

Surgical pathology has a special demonstrator, and the Curator of the anatomical museum, having control of the surgical material obtained at the Massachusetts Hospital, is able to give special instruction in it. In the new buildings now nearing completion, he will have a large room devoted to his department and will have ample accommodation for small classes in this specialty. The surgical department, too, have taken steps towards the establishment of a special laboratory in the Medical School for surgical pathology.

Even this list does not complete the recital of what the Harvard Medical School is doing for

pathology, for at the Bussey Building in West Roxbury the professor of comparative pathology has his laboratory and is constantly at work on the pathological questions suggested by the diseases of animals, and has done much in tracing out their connections with human diseases. The limitations of the laboratory space at his disposal prevents his accommodating students, but his own original researches are making constant valuable contributions to our knowledge of microbic diseases.

In addition to these laboratories and teachers devoted to the study of pathology, the Medical School has also a large and well-equipped laboratory devoted to bacteriology, and a professor with a corps of assistants devoted to the study of the life history of micro-organisms. Besides the instruction and research work done there, great assistance is rendered to private practititioners by the systematic examination of material submitted. Thus this department acts in a measure as a clinical laboratory for the profession outside of the hospitals.

A considerable enlargement of the chemical laboratory and its staff, the establishment of a laboratory for physiological chemistry under able direction, a laboratory of pharmacy and a laboratory of hygiene, with an assistant professor who, besides his research and teaching, gives aid to practitioners by examinations made for them, complete this rehearsal of the present provisions and opportunities for scientific training.

Certainly the contrast between the conditions of twenty-five years ago and those of to-day indicates that our medical teachers are fully awake to and mindful of the demands of modern science. Indeed, despite all the recent improvements that I have imperfectly recounted, the College is at this moment considering a plan for the very great enlargement of the Medical School accommodations. They are driven to this by the need for still more laboratorial space to meet the requirements of modern medical education.

I have selected the Harvard Medical School to serve as an illustration of the recent advances in American methods of study, because it is the institution with which we are most familiar. Similar changes have been going on in greater or less degree in all our centres of medical study, and as a result of this awakening of interest in scientific investigation and teaching, we have already reached the point where it is no longer necessary for American practitioners to go abroad to complete their training in the higher branches of medicine. Ample opportunities now exist in this country for the attainment of an education wholly up to the modern requirements of practice.

It is still wise, for a man who can spare the time, to travel and learn what he can of other points of view and methods of work. I am inclined to think that the time is near, if not already here, when our foreign *confrères* might learn something by coming to this country.

An acquaintance with the literature of a subject

does not widen a man's field of view in the way that an acquaintance with the makers of that literature does.

To return from this digression and to continue our consideration of home matters, we find that this movement towards scientific and accurate methods is not confined to the teachers of medicine. If the time served, it would be a congenial task to say a few words of deserved praise of our Boards of Health.

The executives of State and city have been fortunate in selecting competent, sagacious men to guide the public in matters of sanitation. We are not surprised, therefore, to find that they have been fully alive to the forward movement in medicine, and have established laboratories for the study of sanitary questions. Here are made the many tests necessary in the selection of cases for isolation. Antitoxin is here carefully prepared for distribution, and all possible aid is given to practitioners in the application of laboratory methods to the study of their cases.

We are thus entering the twentieth century with an enthusiasm and a promise for the future which are well justified by what has already been accomplished.

We have many unfinished tasks on our hands. Important principles have been established in the nineteenth century, the detail of which remains to be worked out.

There are many infectious diseases and others believed to be parasitic in origin, of which the microbic causes remain to be discovered.

Our knowledge of the action of microbes in the body and of their effects upon the tissues is in its infancy.

What we know of the action of the tissues in protecting themselves from the micro-organisms and their products is as nothing to what remains to be known.

Efficient antitoxic sera are as yet few, but the list is slowly growing.

These are all questions which bear directly on the successful treatment of disease. We wish, however, to aim higher than this and to learn how to prevent disease.

Our experience with small-pox and diphtheria show that for some diseases, at least, the body may be made resistant by producing alterations in its fluids or tissues. Lister has shown us, too, how other diseases may be prevented by intercepting the microbes and preventing their entrance into the body. This is comparatively easy in surgery, where we have full control of the conditions; can we not protect the body from other infections? Our methods of disinfection of dwellings and public places are constantly improving, but they are still lamentably inadequate. Our boards of health have accomplished something by the isolation of cases of infectious disease and by the extensive use of disinfectants to cleanse the habitations of the sick. The street cars are adorned with signs warning against expectoration, in the hope of thus limiting the spread of tuberculosis, the germs of which are contained so abundantly in the sputa of those afflicted.

May we not hope that further knowledge of the life history of micro-organisms and a more widely spread habit of seeking them out, may make their destruction more generally attempted and more thorough?

If every public hall and school room could be safely and quickly disinfected during the night hours while it is empty, is there any doubt that the public health would be greatly benefited thereby? Sanitary engineering may make this possible, in which case every enlightened community will make it obligatory. Unfortunately there is a force constantly contending against the efforts of our sanitarians, and this is the very steady increase in the numbers and consequent crowding of our populations. For germs flourish in a crowd.

It is to be hoped that the efficiency of our methods of sanitation will be so greatly added to in the near future as to enable us to meet and overcome these increasing difficulties.

There is another nut that the nineteenth century leaves to the twentieth century to crack, if it can. It is the question of the cause and nature of malignant growths. With all the knowledge of them that we have gained through cellular pathology and embryology, we are still left much in the dark with regard to their causation. Bearing considerable resemblance, as some of them do, to processes that are known to have a microbic cause, it is natural that attempts should be made to convict them of a parasitic origin. Without going into the arguments or a minute consideration of the

evidence advanced, I think that I shall be in accord with the best present opinion if I bring in a verdict of not proven.

Diseases of this sort, especially the carcinomata, are apparently on the increase. So prevalent are they and so much to be dreaded that, were it possible to choose in advance the next great medical achievement, the world's vote would probably be for the discovery of a cure for cancer.

It will then perhaps be interesting for you to know that provisions have been recently made in this community looking to systematic research into the cancer question, and I am sure you will be glad to hear what steps are being already taken in this direction. Last year a sum of money was put at the disposal of Harvard College for the investigation of this question. The donor, Mrs. Caroline Brewer Croft, left by will $100,000 for this purpose, and after deducting the exceedingly liberal legacy tax which England exacts, a still considerable sum is left for the furtherance of the wishes of the testatrix. The income of this fund, with some substantial additional gifts obtained by the Professor of Surgery for the same object, are now available for these researches. The investigations under the direction of a committee appointed by the corporation of the college have been put in charge of the department of surgery and the instructor in surgical pathology has been selected to devote himself to a study of the pathological side of the subject. The coöperation of the laboratories of the Boston City Hospital and of

the Massachusetts General Hospital has been obtained and workers in these laboratories have been selected to devote their time to this object. All of these men are in close touch with each other and will have the control of a large amount of material.

At first it is proposed to examine the present theories of cancer production. The question of the relation of germs or parasites to these growths will receive minute and exhaustive attention. Incidental to this examination, the inoculability and infectiousness of malignant growths will be studied.

Besides this study of the pathological side of the subject, attention will be given to the general question of whether the impression that malignant growths are on the increase is well founded. Statistics are already being collected with a view to getting information upon this point, as well as ascertaining the conditions of life which favor the development of these neoplasms and it is hoped that by a long, persistent effort in this direction, light may be thrown on the etiological side of the question.

Something may also be hoped from a patient investigation of remedial agents. In syphilis and in some skin affections we have new growths of tissue which are caused to disappear by the constitutional use of drugs. The effect of arsenic in so-called sarcoma of the skin and the fact that this same drug has, in some cases, effected or seemed to effect the disappearance of a sarcoma which

had invaded the lymph glands, offers some encouragement in this direction.

Such is the beginning of this enterprise. The funds are barely sufficient for a good start, but for so good an object it is to be hoped that others will be forthcoming. In the neighboring state of New York a liberal appropriation has been made by the legislature for similar researches. So much earnest effort cannot be without avail, and it is satisfactory to us all to feel that America is lending her hand to the investigation of this burning question. Whatever comes of it we may be sure the labor will not be wasted.

Most of us here are practitioners of medicine, men so occupied in the care of the sick that but little strength or time is left for the pursuit of purely scientific studies. Are we then of no avail in this forward movement? I do not think so; and I wish to close this rather rambling discourse by pointing out a few of the ways in which the busy profession at large may substantially assist their brethren who are devoting themselves to research.

The mere moral support of feeling that the profession are backing them up and are appreciative of their labors and quick to profit by them, is of great importance to the investigators.

The intelligence of the audience has a stimulating effect upon the teacher, and if we take an active interest in this work and keep ourselves informed of the steps gained, we shall lend the encouragement of our interest to the men work-

ing in the laboratories. We shall, at the same time, fit ourselves to judge properly new ideas and shall be stimulated to observe accurately and record phenomena which pass under our eyes. It must act as an incentive to the men who are working out the higher problems of medicine to feel that any hint they are able to obtain will be at once made of practical value, or will at least be fairly tested and its value determined.

A profession thus made up of men who are in the habit of applying all known scientific methods to the study of their cases and who demand that every patient shall have the advantage of as minute an investigation as is possible, will keep the scientists supplied with material for their studies, and by bringing them in contact with patients will make them in a measure sharers in the emoluments of practice.

This last consideration is no small matter : For in this country, where government does not support its investigators, they must be provided for in other ways. The medical colleges supply places and salaries to a certain number, but there are many men of scientific bent who are unable to follow their inclinations because they must be sure of adequate support for themselves and families. If an enlightened profession demands the services of such men, we may be sure the supply will equal the demand. The army of investigators will not lack recruits. Many a good man will be enabled to spend his life in congenial research instead of seeking his livelihood in the routine of practice, for which perhaps he is ill fitted.

Do not understand me to disparage the practice of healing the sick or to imply that any other pursuit is higher. I only wish to point out that a man, loving the laboratory, may, if driven into general practice, prove to be a square peg in a round hole.

Lastly, it is important that the profession at large, who come in close contact with the people, should keep the community informed of the importance and magnitude of scientific work. In this way only can the proper interest be aroused and necessary endowment and support be obtained.

These are a few of the many duties devolving upon us as we enter the twentieth century. We are in the middle of the stream of medical progress and the current shows no sign of slowing. Let us do our utmost to ensure that it flows on with increasing tide and that there are no back eddies or slack water on the American side.

ARTICLE XIII.

## THE SHATTUCK LECTURE.

# MORBID CONDITIONS CAUSED BY BACILLUS AEROGENES CAPSULATUS.

By WILLIAM H. WELCH, M.D.
OF BALTIMORE, MD.

DELIVERED JUNE 12, 1900.

# MORBID CONDITIONS CAUSED BY BACILLUS AEROGENES CAPSULATUS.

Mr. President and Fellows
of The Massachusetts Medical Society:

Although the subject which I have chosen for this lecture relates for the most part to infrequent affections, the scientific and practical interest attaching to them is considerable and varied. Many instances of the presence of free gas in parts of the body where it does not normally occur and in association with various diseases were recorded by writers of past centuries and were even then the subject of much speculation. The discussion turned generally around the question whether the gas was atmospheric air or the result of putrefaction — a question which in most cases could be solved only by bacteriological examinations. The most numerous and important of such examinations have been made during the last decade, and, although these have left problems still unsolved, they have corrected many current errors and have shed a flood of light upon conditions which were formerly among the most mysterious in pathology.

While it has been demonstrated that various bacteria may be concerned in producing gaseous affections, it is now evident that the bacillus which I discovered in 1891 and to which I gave the name Bacillus aërogenes capsulatus is the one whose causative agency is best established and most frequently in action. What I shall say will relate mainly to this microörganism and its pathogenic effects.

*Historical.*—As a certain amount of confusion concerning the dates of the first publications* on this bacillus exists in foreign literature on this subject, it may be well to state that I reported my observations in November 1891 to the Johns Hopkins Hospital Medical Society and that the full report of these observations and of the characters of the bacillus was published in July–August 1892 by Dr. Nuttall and myself.† E. Frænkel's first publication‡ was a short preliminary one which appeared in January 1893 and was followed in the same year by his valuable monograph on gaseous phlegmons.§ In August 1893, one year after the publication of the paper by Nuttall and myself, appeared simultaneously the interesting articles of P. Ernst‖ and of Graham, Steward and Baldwin.¶ Early in 1894 Mann publised from my laboratory an observation of emphysematous gangrene caused by Bacillus aërogenes capsulatus** and in January 1896 Dr. Flexner and I published an extensive paper reporting twenty-three human cases, including not only six personal observations of emphysematous gangrene but also examples of submucous gas–cysts, pneumoserositis, and various other pathogenic manifestations of this bacillus.†† In July 1895 appeared Goebel's preliminary communication‡‡ and in the following year his full paper on the bacillus of foamy organs.§§ Of the subsequent records the most numerous and valuable have appeared in this country, although they appear to be little known to

* Levy's description in 1891 (Deutsche Ztschr. f. Chirurg., XXXII.) of "kleine, feine" bacilli, cultivated from a gaseous abscess and growing in long threads and chains only at body temperature and cultivable only in the first generation, without animal experiments, cannot be accepted as an identification of B. aërog. capsulatus, or indeed be readily reconciled with its characters.

† Bulletin of The Johns Hopkins Hospital, 1892, III., p. 81.

‡ Centralbl. f. Bakter., XIII., p. 13.

§ Ueber Gasphlegmonen. Hamburg u. Leipzig, 1893.

‖ Virchow's Archiv., CXXXIII., p. 308.

¶ Columbus Med. Journ. XII., p. 55.

** Annals of Surgery, XIX., p. 187.

†† Journal of Experimental Medicine, 1896, I., p. 5.

‡‡ Centralbl. f. allg. Path. u. path. Anat., vi. p. 465.

§§ Jahrb. d. Hamburg. Staatskrankenanstalten, iv.

most European writers.* I shall have occasion to refer later to many of these publications.

*Nomenclature.*—Dr. Fraenkel has kindly favored me with cultures of the bacillus which he cultivated from gaseous phlegmons and to which he gave the name "Bacillus phlegmones emphysematosæ." There can be no question whatever as to the identity of his bacillus with our Bacillus aërogenes capsulatus, a point upon which we are both agreed, and which is also made certain by Goebel's studies under Fraenkel's supervision. According to the generally accepted principles of the nomenclature of zoological and botanical species the latter name, as being the first one applied, should be preferred to the former. It is, moreover, as pointed out by Muscatello, not open to the objection of implying exclusive relationship to a single disease, as is the case with Fraenkel's designation of the bacillus. As a matter of fact the capacity to produce gaseous phlegmons is only one of many pathogenic manifestations of Bacillus aërogenes capsulatus. Unfortunately I think that we both erred against the canons of botanical nomenclature in using a trinomial rather than a binomial name for a species.†

*Characters of Bacillus.*—Since our first publication only one material addition has been made to the extended

* Thus v. Hibler in 1899 (Centralbl. f. Bakt. XXV., p. 513 et seq.) in an elaborate study of pathogenic anaërobes is entirely ignorant of our work and that of other American investigators on B. aërog. capsulatus. The information of Hitzschmann and Lindenthal (Sitzungsb. d. K. Akad. d. Wiss., Math.-Naturw. Cl., Wien., 1899,) on the American work is secondhand and both incomplete and inaccurate, in these respects being in unfavorable contrast to that of Muscatello and Gangitano (Riforma Med., 1900, II. p. 508, et seq.) who write also on the subject of emphysematous gangrene. Knowledge of Bacillus aërogenes capsulatus, under the name Bacillus perfringens, has begun to appear in France in the last two years, but without any evidence of acquaintance with the American publications.

† Migula, who, with considerable success, has attempted to reform bacteriological nomenclature, has given the binomial names "Bacterium Welchii" to Bacillus aërogenes capsulatus and "Bacterium emphysematosum" to Bacillus phlegmones emphysematosæ (System der Bakterien, II., pp. 392 and 383). He is, however, in error in describing this organism under two different names, as his Bacterium Welchii and Bacterium emphysematosum are identical.

description given by Nuttall and myself of the morphological and cultural characters of Bacillus aërogenes capsulatus or the gas bacillus, as I shall briefly call it.* Fraenkel noted the presence of spores in a few of the bacilli growing in one lot of agar containing sodium formate, and in 1897 Dunham† observed spores in blood-serum cultures, but not in other media.

Our further studies of the gas bacillus obtained from different sources have shown a moderate range of variation in some of its properties. This is true especially of spore-formation, rapidity of liquefaction of gelatine, presence of capsules, retention of Gram's stain, and virulence. While some specimens of the bacillus seem never to form spores on any culture-medium, others, and these appear to be the more common, do so occasionally, especially upon blood-serum, in mannite bouillon, and on plain agar. In animals inoculated with pure cultures we have not observed spore-bearing bacilli.

As a rule the bacillus liquefies gelatine slowly, but some specimens do so scarcely at all, and others with fair rapidity.‡

As stated in our original article capsules are not constantly present, but I have generally found no difficulty in demonstrating them in the situations and by the method described by us, and, with the exception of Hitschmann

* As regards these characters, it will suffice here to say that the microörganism is a rather coarse, non-motile, anaërobic bacillus, staining by Gram, growing on all of the ordinary culture media under anaërobic conditions, best at body temperature, but also at room temperature, forming spores inconstantly according to the race and the culture-medium, and capable of forming gas, not only by fermentation of sugars, but also from proteids. The full description of the characters may be found in Welch and Nuttall's paper. The gas, according to Dunham's analyses, is composed approximately of 64 per cent. hydrogen, 28 per cent. carbon dioxide, and 8 per cent. of a residual gas believed to be mainly nitrogen. It has no foul odor.

† Bulletin of The Johns Hopkins Hospital, 1897, VIII., p. 68.

‡ In our original communication we noted peptonization and softening of the gelatine, but this was so slow and slight that we then preferred to class the bacillus among the non-liquefiers. Further experience has shown that the bacillus is a liquefier, but generally a slow one.

and Lindenthal, most other investigators have been able to demonstrate capsules, when these are searched for by suitable methods.

While the bacillus is to be ranked among those which stain by Gram, it is sometimes rather noticeable in coverslips from cultures that among well stained bacilli others are partly or wholly decolorized, and this may be observed in members of a single chain. In the tissues the bacillus stains well by Gram. Differences in the viability of cultures were pointed out in our first paper.

It sometimes happens that original cultures from human beings show only a feeble growth, with relatively weak power of gas-production, while subsequent cultures, especially those obtained after passage through the animal body, present the usual vigorous growth and other typical characters. Pratt and Fulton* from a typical case of foamy organs, with gas throughout the body, were unable to cultivate the bacillus at all, although twelve anaërobic culture tubes containing various media were inoculated from different parts of the body. This negative result they attribute to the fact that the body had lain in a cold storage vault for sixteen hours after death, an explanation which has some support in an observation previously reported by Welch and Flexner, but still seems hardly satisfactory, as under similar conditions the bacillus has been often cultivated.

Lactose, glucose and saccharose are all fermented by the gas bacillus, the first with the largest production of gas and the last with the smallest. There is apparently no fermentation of mannite, at least the gas is not appreciably more than in sugar-free media. The amount of hydrogen greatly preponderates over that of carbon dioxide.

E. Fraenkel in 1893 was the first to demonstrate the etiological relation of the gas bacillus to gaseous phlegmons, our previous investigations being concerned mainly

* Boston Med. and Surg. Journ., June 7, 1900, p. 599.

with the so-called foamy organs (Schaumorgane) and the presence of gas in the blood, our results being confirmed a year later by P. Ernst. Soon after Fraenkel's publication we were able to confirm his discovery of the causation of gaseous phlegmons by Bacillus aërogenes capsulatus, and to repeat with similar results his animal experiments.

Material or cultures fresh from the infected body are usually highly virulent for guinea pigs, pigeons and sparrows (E. Fraenkel), which succumb to a rapidly spreading local necrosis of the tissues with abundant development of gas, the bacilli invading the blood during life only in small number or not at all. There is more or less bloody œdema, but otherwise little inflammatory reaction, leucocytes being present usually only in small numbers in the exudate. Rabbits and mice, while not wholly immune, are far less susceptible than guinea pigs and pigeons. Dr. Lanier in my laboratory in 1896 succeeded in producing typical gaseous phlegmons around the fractured bones of rabbits inoculated intravenously with pure cultures and Muscatello has obtained the same results. There are considerable differences in the degree of virulence of the bacillus even in fresh cultures, and old ones may be of very slight virulence.

One of the most interesting and valuable tests of the gas bacillus is its power of producing gas abundantly in the blood, organs and tissues of rabbits killed a few minutes after intravenous injection, a power not possessed by colon bacilli. The differential value of this test is as great as that of cultures in fermentation tubes. The blood and tissues of the dead rabbit make the culture medium, the body of the animal takes the place of the test tube, the inoculation is an aseptic one, the bacteria are spread by the blood-current and the conditions are anaërobic. This procedure, which was introduced by Nuttall and myself and has been fully described by us, we have found useful in isolating the bacillus, in separating it from other bacteria

which may resemble it, and in the demonstration of one of its most fundamental characteristics, namely, the power to produce gas from proteid material.

Among the points distinguishing the bacillus of malignant œdema from Bacillus aërogenes capsulatus may be mentioned the following: The malignant œdema bacillus is somewhat thinner, has greater tendency to grow into filaments, is less readily stained by Gram, produces spores regularly in culture media, is motile, liquefies gelatine more rapidly, produces a foul odor, produces less gas in lactose bouillon, peptonizes clotted casein, generates little or no gas in rabbits inoculated intravenously and then killed, and by subcutaneous inoculation in susceptible animals causes spreading bloody œdema with little or no development of gas bubbles, and appears after death in filaments on serous surfaces.

The bacillus to which Lindenthal* has quite unnecessarily given the name Bacillus emphysematis vaginæ is doubtless identical with Bacillus aërogenes capsulatus. The same is true of Veillon and Zuber's† Bacillus perfringens, found by them in appendicitis, by Guillemot‡ in gaseous gangrene and by Soupault and Guillemot§ in gaseous abscesses, and of Buday's‖ Bacillus cadaveris butyricus, found in foamy organs. I am strongly inclined to the opinion that the anaërobic bacillus isolated by Achalme and others from several cases of acute articular rheumatism and found by Savtchenko and Mielkich in the soil is likewise identical with our gas bacillus.¶

*Distribution.*—The surmise expressed by Welch and Nuttall that the gas bacillus is widely distributed in nature

* Wiener klin. Wochensch, 1897, p. 3, et seq.
† Arch. de méd. exp., 1898, X. p. 539.
‡ Compt. rend. Soc. de biol. 1898, 10. s., v, p. 1017.
§ Bull. et mém. Soc. méd. d. hôp. de Paris, 1900, 3. s, xvii, p. 216.
‖ Centralbl. f. Bakt., 1898, xxiv, p. 369.
¶ Achalme, Ann. de l'Inst. Pasteur, 1897, XI. p. 845; Pic and Lesieur, Journ. de phys. et de path. gén., 1899, I., p. 1007, and Savtchenko and Mielkich. Arch. russes de path., 1899, VIII. p. 145.

has since been confirmed. The natural habitat of the organism is the intestinal canal and the soil, the home of so many other anaërobic bacteria. Welch and Flexner in 1896 brought evidence of the presence of the bacillus in both of the situations mentioned. Clopton of the Johns Hopkins Hospital has found the bacillus twice in the appendix vermiformis. Howard* has recently reported the presence of morphologically identical bacilli in the intestines of twenty-five consecutive human cases examined post mortem, and in ten of these he demonstrated the bacillus by cultures and inoculation of animals. The same conclusion has been reached by Hitschmann and Lindenthal. The gas bacillus has been repeatedly cultivated from the intestine in my laboratory, but we have made no systematic study of the frequency of its presence. I have found the bacillus also in the intestines of rabbits, dogs and swine, and here it is interesting to note the frequency with which submucous gaseous blebs are found in the pig's intestines at autopsy.

In 1896 Dr. Walker, at the Johns Hopkins Hospital, succeeded in finding the gas bacillus in dust collected by sweeping floors, proving its presence both by cultures and animal experiments. My assistant, Dr. Harris, has cultivated the bacillus from the contents of an old cesspool. I had previously reported in 1896 the isolation of the bacillus from a bullet removed from the head of the tibia in a case of gaseous phlegmon, and E. Fraenkel† has cultivated the gas bacillus from a splinter of wood extracted from a wound in a case of tetanus. These observations confirm the natural inference to be drawn from the study of cases of traumatic emphysematous gangrene, in most of which the source of infection is manifestly foreign material, especially dirt, in wounds. In the light of these demonstrations of

* Contributions to the Science of Medicine dedicated by his pupils to William Henry Welch, on the 25th Anniversary of his Doctorate, p. 461, Baltimore, 1900.

† Münch. med. Woch., 1899, No. 42 and 43.

the wide distribution of the gas bacillus in the outer world and in fæces, the conclusion is warranted that it must occasionally be present upon the human skin.

We are not informed whether there are differences in the regional distribution of the gas bacillus. The fact that during the last decade a larger number of cases of emphysematous gangrene have been reported from Baltimore than from any other single locality is due probably to our interest in the subject and consequent search for cases. The bacillus has been found not only in America and Europe, but Dr. Flexner has brought back reports of three infections with the gas bacillus in Manila, observed during a stay of three months.

### GAS BUBBLES IN THE BLOOD AND ORGANS.

We turn now to the consideration of the various conditions in which the gas bacillus has been found in human beings. We need not pause to consider the presence of this bacillus in ordinary cadaveric decomposition, a circumstance sufficiently explicable by the occurrence of this organism in the healthy intestinal canal.

Of an entirely different nature are the cases in which gas bubbles are found in the blood and organs within a few hours after death and without any trace of ordinary putrefaction. Such a condition has been recognized at autopsies as soon as one, two, three, five, eight hours after death. It may occur not only after death from gaseous phlegmon, when, however, it is by no means constant, but also after death from the most varied causes. It has been observed repeatedly in autopsies on pregnant and puerperal women, especially after death from abortion and acute sepsis.

There is every gradation from cases with a few bubbles of gas in the blood or tissues to those with extensive emphysema of the organs and tissues. The term "foamy organs" (Schaumorgane of the Germans) may be applied to the

latter condition. The liver is the organ most frequently the seat of early and abundant development of gas, but there is no definite rule as to the distribution and amount of gas in different cases. As will be explained later, the invasion in the majority of cases is from the intestine. That the gas bubbles may be dislocated from their original position in liquid and soft material in the body is self-evident, but I have not found them unassociated with gas bacilli.

Formerly this early presence of free gas in the heart and vessels, without evident post-mortem decomposition, was very generally explained by the assumption of entrance of air into the circulation, even when no portal of entry could be found.* The most extensive application of this explanation was made in the pregnant and puerperal cases. It is remarkable that the first case of this nature to be examined bacteriologically was that reported by me in 1891. In this and in all subsequent similar cases with satisfactory bacteriological examination Bacillus aërogenes capsulatus was found.

The main questions which arise concerning the interpretation of these cases are whether the invasion of the bacilli and whether the development of the gas are ante-mortem or post-mortem phenomena.

Rabbits survive the introduction of large numbers of gas bacilli directly into the circulation, unless there exists somewhere in the body necrotic or damaged tissue offering little or no vital resistance. If the animal be killed within a few minutes after the intravenous injection of the bacilli and kept in a warm place, there are abundant multiplication of the bacilli and large development of gas throughout the body within the space of six or eight hours; whereas if the bacilli be introduced at one point, as for example the right heart, of a rabbit just killed, it takes a much longer time,

* Cless. Luft im Blute. Stuttgart, 1854, and Couty. Thèse, Paris, 1875.

often twenty-four or forty-eight hours, for gas and bacilli to make their appearance at points far distant from the seat of inoculation. It seems justifiable to draw from these three groups of experiments, which have been fully reported by Welch and Nuttall, the conclusion that when bacilli and gas are found within a few hours after death widely distributed in the body, the gas bacilli have entered the circulation during life, but probably in most cases only shortly before death.

There is one factor, however, to be considered which is absent in the experimental cases and may be present in human beings, to wit, the quick disappearance of the bactericidal power of the blood. This factor is an important determinant of the rapidity of onset of post-mortem decomposition. Very soon after death from certain diseases, and particularly from snake-venom, bacteria may make their appearance in the blood and organs. An explanation of these cases is furnished by the experiments of Ewing and myself,* which demonstrated that the blood of rabbits killed by rattlesnake venom is practically devoid of bactericidal power so that immediately after or even shortly before death bacteria can start growing in the body as they would in a tube of beef-broth. But after all due allowance has been made for the possible reduction or loss of bactericidal power of the blood, I still consider that it is not possible to explain some of the cases in which bacilli and gas have been found in the heart, blood-vessels and organs very soon after death, especially when the corpse has been kept in a cold place, otherwise than upon the assumption of the distribution of the bacilli by the circulating blood.

It is another question whether gas as well as bacilli may be present in the circulating blood and internal organs during life in the class of cases now under consideration, and I

* Lancet, 1894, I. p. 1236.

regret to be unable to furnish a positive answer to this question. I do not see how an affirmative answer can be obtained otherwise than by the actual demonstration of gas in these situations either during life or immediately after death. Gas bubbles and bacilli have been found in the heart and vessels within an hour after death, but that is time enough for bacilli which have already been introduced to multiply and begin to form gas. I at first thought that absence of nuclear staining around the gas bubbles and masses of bacilli might serve as an indication of their presence during life, and this view is advocated by P. Ernst, but I have since learned from experiments on rabbits that this is not a decisive criterion, although often both in rabbits and in human beings there is no defect in nuclear staining around the bacilli and gas bubbles.

I know of no other pathogenic micro-organism which offers such difficulties in determining whether its effects in the interior of the body have been produced before or after death. The difficulty arises from the circumstance that these effects in most cases and most situations consist almost entirely in local necrosis and formation of gas, whether the invasion and growth of the bacilli be before or after death, and that unlike most pathogenic bacteria the gas bacillus grows better in the dead than the living body. Possibly some importance in the solution of the problem may attach to the demonstration of emboli of liver cells and of bone-marrow cells which were in enormous numbers in the pulmonary vessels in a case of gaseous phlegmon of the submammary tissues following infusion of salt-solution. At the autopsy made by Dr. Carroll the liver and other organs were emphysematous. Further observations with reference to these emboli in this class of cases are needed.

I do not consider that there is any inherent improbability in the supposition that gas bubbles may be in the circulating blood during life without causing speedy death from

gaseous embolism. It is only when a large volume of air is introduced quickly into the blood-current that sudden death results from air embolism. Very exaggerated ideas have prevailed among physicians as to the dangers from the entrance into the circulation of small quantities of air. Laborde and Muron* injected into the external jugular vein of a dog 1120 cc. of air in the space of one hour and a half without causing death, and Jürgensen† into the left femoral artery of a narcotized dog weighing 43½ kilo 3,650 cc. in the space of two hours and twenty-five minutes with only slight disturbance of the respiration and of the action of the heart. Hare‡ on the basis of experiments likewise controverts current beliefs concerning the dangers from entrance of air into the veins.

I have come across in the older literature, from the days when venesection was a common practice, reports of cases in which blood containing bubbles of gas escaped during venesection from veins of the arm.§ In none of these was there evidence that air had gained entrance to the circulation. Maisonneuve,‖ in incising two gaseous phlegmons of the thigh following compound fracture, observed the escape of blood containing gas-bubbles from the cut veins, and was able to trace the gas within the veins for a long distance.

It seems to me very improbable that an anaërobic bacillus, such as the gas bacillus, can multiply in the circulating blood, still this bacillus is less sensitive to the presence of oxygen than many anaërobes, and we do not know whether the loose combination in which oxygen is present in the blood would necessarily prevent its growth under all circumstances.

I see no reason why this bacillus might not multiply in the liver, spleen and most other internal organs, as we

* Comptes rend. Soc. de la biol., 1873, V.
† Deutsches Arch. f. kl. Med., 1882, XXXI., p. 458.
‡ Therap. Gazette, 1889, 3. s., V. p. 606.
§ Marshall's case reported by May, Trans. Path. Soc., London, 1858, IX., p. 157; Durand-Fardel's case also cited by May, and Pirogoff's case in his Grundzüge d. allgem. Kriegschirgie, p. 1063, Leipzig, 1864.
‖ Cited from Hitschmann and Lindenthal, loc. cit.

know it can do in parts open to inspection during life. We have positive evidence in the cases reported by Graham, Steward and Baldwin, and by Dunham that gas bacilli may be conveyed by the circulation from an infected portal of entry, in the one case the puerperal uterus and in the other a urethral wound, to distant parts of the body, and there produce subcutaneous emphysema and necrosis. There is no part of the body which offers such favorable conditions for the post-mortem growth of the bacillus as the liver, probably on account of its content of carbohydrate, and, if the liver like the integuments were open to inspection during life, I believe that we should find evidence that in certain cases the emphysema of this organ, which is such a conspicuous post-mortem phenomenon in instances of invasion by the gas bacillus, had begun during the life of the patient. Emphysema of mucous membranes, open to inspection, we know can exist during life. This subject will be considered subsequently.

In the great majority of the instances, however, in which gas-bubbles are found in the blood and internal organs at autopsy, the evidence is in support of the view that the development of the gas is a purely post-mortem phenomenon. Certainly the greatest caution should be exercised in the interpretation of any such cases as vital processes, even in early autopsies without ordinary putrefaction. One thing which our investigations have established is that the finding of gas-bubbles in the blood-vessels and heart within as short a time as one hour after death, furnishes in itself no proof of the entrance of air into the circulation. I shall refer later to the question of gaseous embolism in cases of emphysematous gangrene and of physometra.

### EMPHYSEMATOUS GANGRENE.

In a few instances we have found in wounds, usually compound fractures or gunshot injuries, in which dirt had

gotten in, Bacillus aërogenes capsulatus without the presence of gas or other evidence that the bacillus was producing any characteristic effects.* Such cases have always been watched by the surgeons with anxiety, and it is probable that at least in some the early recognition of the bacillus, followed by free incisions and thorough cleansing and disinfection, has warded off a subsequent grave infection. In view of the wide distribution of the gas bacillus in the outer world and in the intestinal contents, it is probable that it must not so very infrequently gain access to wounds without securing a foothold. While this innocent behavior, with which we are also familiar in the case of the tetanus bacillus, may sometimes be due to attenuated virulence of the bacillus, it is probably oftener attributable to accessory circumstances, such as the resistance of the patient, the condition of the wound and surrounding tissues, and association with other micro-organisms and with foreign substances.

It is as a cause of that most dreaded of wound complications, emphysematous gangrene, that Bacillus aërogenes capsulatus especially claims the interest of surgeons. The classical clinical descriptions of this disease we owe to Maisonneuve† and to Pirogoff,‡ the former giving to it the name "gangrène foudroyante," and the latter designating it "primary mephitic gangrene" or "acute gangrenous œdema." Among other more or less common designations are "emphysematous or gaseous gangrene," "gaseous phlegmon," "septic emphysema," "érysipèle bronzé" (Velpeau), "progressive gangrenous œdema," "gangrenous septicæmia," and "emphysematous cellulitis."

This wound complication was more common in preantiseptic times, especially in military surgery, than it is to-day; but at least seventy cases have been reported dur-

* Such cases have been reported by Bloodgood of the Johns Hopkins Hospital in Progressive Medicine, December, 1899, IV., p. 158.

† Gaz. méd. de Paris, 1853, p. 592.

‡ Grundzüge d. allgem. Kriegschirurgie, pp. 867 and 1006, Leipzig, 1864.

ing the last quarter of a century. In prebacterial days the affection was attributed by some writers to the penetration of air into the tissues, but by most to the decomposition of the tissues, particularly of adipose tissue and bone-marrow, brought by an injury into contact with the atmosphere.

Bottini,* in 1871, was the first to demonstrate the infective nature and transmissibility of emphysematous gangrene. Later, Gussenbauer also recognized the disease as a definite infection, and attributed it to the bacteria of putrefaction. After Pasteur's discovery in 1877 of his "vibrion septique," more commonly, since Koch and Gaffky's investigations, designated Bacillus œdematis maligni, and especially after Chauveau and Arloing's† paper in 1884, cases of emphysematous gangrene have been usually reported, especially in France, as instances of Pasteur's gangrenous septicæmia or Koch's malignant œdema. W. Koch's‡ attempt to identify the disease with symptomatic anthrax (Rauschbrand) was based on faulty bacteriological studies and has met with no confirmation. In 1884 F. J. Rosenbach§ reported finding in cover-slip specimens from two cases of traumatic emphysematous gangrene coarse bacilli, some of which had terminal spores. These he was unable to cultivate, only aërobic methods being employed. It is probable that Rosenbach saw the gas bacillus in these cases, but without distinguishing it from associated spore-bearing bacilli.

A critical examination of the records of alleged malignant œdema in human beings shows that in very few was the organism concerned satisfactorily identified as the genuine malignant œdema bacillus. Very often it has been simply assumed without more than a microscopical examination that bacilli found in spreading œdematous conditions with

*Gior. d. r. Accad. di med. di Torino, 1871, 3. S., X., pp. 1121 and 1138.
† Bull. Acad. de Méd. 1884, 2. S., XIII., p. 604.
‡ Deutsche Chirurgie, Lief. 9, Stuttgart, 1886.
§ Die Mikro-organismen bei den Wund-Infections-Krankheiten des Menschen, Wiesbaden, 1884.

or without gas have been those of malignant œdema, and even where cultures and animal experiments have been employed the descriptions are frequently so meagre as to leave the identity of the organism wholly in doubt. In France it is usually assumed without any discussion and even without any bacteriological examination that gangrène foudroyante is malignant œdema (Pasteur's septicæmia),* and the same ignorance of the present status of this subject is still sometimes encountered in England, Germany and elsewhere. Nevertheless the investigations of the last seven years, beginning with those of E. Fraenkel and soon followed by observations of myself and collaborators, have demonstrated that by far the most common and important specific cause of gaseous phlegmons or emphysematous gangrene is Bacillus aërogenes capsulatus.

Whether the bacillus of malignant œdema can produce an identical or similar anatomical and clinical affection in human beings I regard as an unsettled question. It is certainly remarkable in view of current doctrines in text-books that neither E. Fraenkel nor I with our relatively large experience, nor indeed, so far as I am aware, anyone who has made himself thoroughly acquainted with Bacillus aërogenes capsulatus, has encountered an instance of emphysematous gangrene in man caused by the bacillus of malignant œdema. The whole subject of human malignant œdema is one which needs thorough revision and investigation by more exact bacteriological methods than have yet been applied to it.† I have already mentioned the chief points

* An exception is Guillemot (Comptes rend. Soc. de biol., 1898, 10. s., v. p. 1017) who has found B. aerog. caps. in a case of gaseous gangrene and who controverts the prevalent view of authors who attribute this disease exclusively to Pasteur's vibrio.

† In the case reported recently by Brabec (Wiener klin. Rundschau, 1900, XIV., pp. 145 and 167) the identification of the malignant œdema bacillus seems satisfactory. Here there was extensive bloody œdema without gas, so that the case was not one of emphysematous gangrene. On the other hand Hämig and Silberschmidt (Correspondenzbl. f. Schweizer Aerzte, 1900, XXX., p. 361) bring no proof of any consequence that they were dealing, as they supposed, with the malignant œdema bacillus in two cases of gangrène foudroyante.

of difference between the bacillus of malignant œdema and Bacillus aërogenes capsulatus.

There is a relatively small group of cases of gaseous phlegmon attributed by those reporting them* either to the colon bacillus or the proteus bacillus. In most of these cases anaërobic culture methods were not employed. No one has succeeded in producing experimentally gaseous phlegmon with either of these bacilli, and I think there is good reason to be skeptical concerning their capacity to produce this disease, unless perhaps Bacillus coli may do so in diabetics.

It is possible that some of those reporting the colon bacillus as the cause of emphysematous gangrene may have confounded with it a facultative anaërobic bacillus which we have isolated from two cases of this disease, and which has been studied in my laboratory by Dr. Lanier. It resembles in anaërobic cultures very closely Bacillus aërogenes capsulatus, but it is capable of aërobic growth also, and then the rods are thinner and more like colon bacilli. It has the power of producing gas abundantly in the blood and tissues of rabbits killed a few minutes after intravenous injection, a power not possessed by genuine colon bacilli. I have already spoken of the importance of this test, which has been employed by none of the writers who have claimed to find colon bacilli as the cause of gaseous phlegmons. This bacillus, when virulent, is capable of causing the same spreading and fatal emphysematous necrosis in guinea pigs and pigeons as is Bacillus aërogenes capsulatus.

I have been accustomed to speak of this bacillus, to which I have called attention in previous publications, as the aërobic variety of our gas bacillus. I believe, however, that it is identical with Sanfelice's Bacillus pseudo-œdematis maligni† with which he is inclined to iden-

* Chiari, v. Dungern, Bunge, Klemm, Hlava, Evans, Grasberger, Hauser, Margarucci, Muscatello, Hitschmann and Lindenthal.

† Ann. d. Istit. d'Igiene sper. d. Univ. di Roma, 1891, N. S., I., p. 365, and Ztschr. f. Hyg. 1893, XIV., p. 352.

tify Klein's "new bacillus of malignant œdema."* Chavigny† has isolated apparently the same bacillus, which he likewise identifies with Sanfelice's Bacillus pseudo-œdematis maligni, (not to be confounded with the pseudo-œdema bacillus of Liborius), from a case of gaseous gangrene, and he also calls attention to the probability that others may have mistaken it for the colon bacillus. While, therefore, unwilling upon existing evidence to accept the colon bacillus as a demonstrated cause of gaseous gangrene (except perhaps in diabetics), I am of the opinion that an aërobic bacillus, probably identical with Sanfelice's Bacillus pseudo-œdematis maligni, is capable of producing this affection, but it is much less frequently concerned than Bacillus aërogenes capsulatus.

I have collected forty-six cases of emphysematous gangrene, in all of which Bacillus aërogenes capsulatus was demonstrated, and, therefore, all reported or observed during the last seven years.‡ This is a far larger number of cases than has ever been brought together before.

* Centralbl. f. Bakter., 1891, X., p. 186.

† Ann. de l'Inst. Pasteur, 1897, XI., p. 860.

‡ This list includes 16 cases observed in Baltimore, mostly at the Johns Hopkins Hospital, of which 2 are unpublished, and the remaining 14 have been published by Mann (1), Ann. Surg., 1894, XIX., p. 187; Welch and Flexner (6), Journ. Exp. Med., 1896, I., p. 5; Martin (1), University Bulletin, 1896, I., No. 3, and Bloodgood (6), Progressive Medicine, 1899, IV., December, p. 158. The notes of an additional unpublished case observed in Manila have been given me by Dr. Flexner. There are also 3 unpublished cases for the records of which I am indebted to Dr. Carroll of Washington.

The references to the remaining 26 cases are as follows: E. Fraenkel (4), Ueber Gasphlegmonen, Hamburg u. Leipzig, 1893; Passow (1), Charité-Annalen, 1895, XX., p. 275; Dunham (5), Bulletin of Johns Hopkins Hospital, 1897, VIII., p. 68; Ferguson (1), Trans. Indiana Med. Soc., 1897, p. 339: Erdmann (1), Med. Record, Feb. 5, 1898, p. 205; Le Boutillier (1), Med. Record, March 5, 1898, p. 353; Love and Cary (1), Med. Record, April 8, 1899; Norris (1), Amer. Journ. Med. Sc., 1899, CXVII., p. 195; Hitschmann and Lindenthal (5), Sitzungsb. d. k. Akad. d. Wiss., Math-Naturw. Cl., Wien, 1899, CVIII., Heft. iii., Abth. iii., p. 67: Thorndike (2), Boston Med. and Surg. Journal., June 7, 1900, p. 592; Muscatello with Gangitano (3), Riforma med., 1900, II., pp. 508, 519 and 530; Guillemot (1), Compt. rend. Soc. de biol., 1898, 10. s, V. p. 1017. A few other cases in which the gas bacillus was found are not reported with sufficient detail to be available for analysis. It is safe to say that B. aërog. caps. has now been found in over 50 cases of gaseous gangrene.

Thirty-two are reported by American observers and only fourteen by foreign investigators. Of the former group of cases sixteen were observed in Baltimore, most of the cultures having been studied in my laboratory; of the foreign group of cases all are reported from Germany, Austria, Italy and France, four by E. Fraenkel, one (not absolutely certain) by Passow, five by Hitschmann and Lindenthal, three by Muscatello assisted by Gangitano, and one by Guillemot.*

Cases of gaseous phlegmon in which Bacillus aërogenes capsulatus was not demonstrated are not included, although many of these presented the same clinical characters and doubtless in some at least the gas-bacillus was the active agent. This is true especially of the cases of gangrène foudroyante, usually without satisfactory bacteriological examination, attributed by French writers to Pasteur's vibrion septique. Gertler's† eight cases of gaseous phlegmon cannot be utilized for our purposes at all, as they are without any satisfactory bacteriological reports.

A complete analysis of these 46 cases would afford material more than sufficient to occupy this entire address, so that I shall be able to present here only some of the more important points.

Thirty-five of the patients were males, ten females, and of one the sex is not stated. The preponderance of males is to be explained by the fact that most of the cases were due to severe injuries. Robust workmen in the prime of life furnished the largest contingent of cases.

In 80 per cent. of the cases one of the extremities was the seat of the emphysematous gangrene, the lower being

* Soupault and Guillemot (Bull. et mém. Soc. méd. d. hôp. de Paris, 1900, 3. s., XVII., p. 216) have reported two gaseous abscesses following hypodermic injections of salt solution, and a third which they regard as metastatic, in which they found the gas bacillus (B. perfringens), once in pure culture. These cases came to my notice too late to be included in my statistics. All were relatively benign cases.

† Ueber Gasphlegmonen. Inaug.-Diss. Halle, 1898.

affected a little over twice as often as the upper extremities. In several instances the emphysema extended from the thigh to the abdominal wall or from the arm to the subcutaneous tissues of the shoulder and chest. There were three examples of primary emphysematous phlegmon of the abdominal wall, of these one following removal of the appendix (Bloodgood), one from an unrecognized strangulated Littré hernia (Martin), and one affecting the deep tissues of a nephrectomy wound (Muscatello). In all of these the infection is believed to have started from the intestine. In one of Dunham's cases there was emphysematous gangrene (originating in a prostatic abscess opening in the buttock) of the scrotum, penis, and anterior abdominal and thoracic subcutaneous tissues. In three instances (Carroll (2), Dobbin (1) reported by Bloodgood) the breast and submammary tissues were the primary seat of the disease, all of these resulting from the infusion of normal salt solution. In one case (Dunham) the gaseous phlegmon appeared at the angle of the lower jaw after incision of a foul submaxillary abscess. In one instance (Welch and Flexner) it started within the pelvis from traumatic rupture of the rectum and extended through the sciatic notch down the thigh.

Of especial interest are three examples of multiple or metastatic emphysematous gangrene: one of the forearm and opposite shoulder, another of the thigh and both shoulders, and still another of one shoulder and the buttocks. In the older literature are similar cases; thus Nelaton observed emphysema not only in the injured leg but also in the opposite, uninjured extremity. In 1897 Leech* reported, without adequate bacteriological examination, a case of emphysematous gangrene of the right leg following about three weeks after injury of the right thumb, which became inflamed, there being no evident local cause of the

* Quart. Med. Journ. (Sheffield), 1896-97, V. p. 237.

affection of the leg. These cases are to be explained by transportation of the bacilli through the lymphatic or blood current from the primary focus of entrance.

In all but five of the forty-six cases the emphysematous gangrene followed traumatism or a surgical operation. The injuries were as follows: compound fractures, 18; bullet and gunshot wounds, 7; infusion of salt solution, 3; hypodermic injections, 2; ligation of the femoral artery for aneurism, 3; external urethrotomy, 2; traumatic rupture of the rectum, removal of the appendix, prostatic abscess following self-catheterization, operation for strangulated Littré hernia, incision of a foul submaxillary abscess, and nephrectomy, each one. Of the five non-traumatic cases the gaseous gangrene followed erysipelas in one, was consecutive to apparently spontaneous gangrene in two, whether diabetic or not is not stated, and was without apparent explanation in two (Fraenkel's case 2 and Passow).

Compound fractures and next bullet and gunshot wounds occupy by far the most prominent place in this list, each of the other various causes being represented only by scattered cases. Those injuries, in which there are much laceration and crushing of tissue, comminution of bone and grinding of dirt, bits of clothing or other foreign bodies into the wound are the ones most likely to be followed by emphysematous gangrene. That, however, severe traumatism is not an essential factor is shown by the five cases (10.9 per cent. of the entire number) following hypodermic injections and infusion of normal salt solution. These latter, however, were all of patients whose vital forces were greatly depressed, namely, by Asiatic cholera, typhoid fever, surgical shock, or post-partum hemorrhage. There is good reason to believe that the intact tissues of human beings in health possess marked resistance to the gas bacillus.

In the great majority of cases it was evident that the gas bacillus was introduced through the wounded skin from

without. In three instances (removal of appendix, strangulated hernia, and traumatic rupture of the rectum) the infection undoubtedly came from the intestine. This was also the probable source of infection in one of Muscatello's cases (gaseous phlegmon in the site of an extirpated kidney). With our present knowledge of the frequent, if not regular, presence of Bacillus aërogenes capsulatus in the intestine, there is nothing surprising in this mode of infection. In three of Dunham's cases the infection followed injury of the urethra, and here also the bacilli may have come from the intestine. In one of his cases (gaseous phlegmon at angle of the jaw) it was suggested that the bacilli gained entrance through decayed teeth.

Bloodgood thinks it probable that in one of his cases the gas bacilli were brought by the circulation to the seat of infection. In this case the femoral artery was ligated for traumatic arterio-venous aneurism in the popliteal space. There was no primary infection of the wound, but gangrene of the foot and leg ensued, and on incision gas bubbles were found in the blood of the aneurismal sac and the tissues. With what we know about the entrance of intestinal bacteria into the circulation, there is nothing improbable in Bloodgood's opinion. The clot in an aneurism and tissues robbed of their nutrient supply would offer little or no resistance to the growth of bacilli which might reach them or their immediate neighborhood through the circulation, and the conditions would be anaërobic. It was indeed a case of aneurism in which I first found the gas bacillus, and here the clot was swarming with bacilli. It is interesting to note that three of the cases of emphysematous gangrene in my list followed ligation of the femoral or popliteal artery for aneurism.

Pirogoff* distinguished clinically two groups of cases of traumatic emphysematous gangrene. He described under

* Op. cit. p. 1006.

the name "primary mephitic gangrene" cases in which the emphysema appears within two days after the injury, the "local stupor" passing without inflammatory reaction into crepitating gangrene. Here the emphysematous necrosis spreads rapidly, the patient sinks into collapse with an icteroid hue of the skin, small, thready pulse and cold sweats, and death occurs usually within a few days after the onset. This type of the disease corresponds to Maisonneuve's gangrène foudroyante. In the second group, designated by Pirogoff also as "acute gangrenous œdema," there is reaction from the primary "local stupor" of the tissues, the emphysema is preceded and accompanied by local, œdematous or purulent inflammation, is associated with febrile reaction, often appears later after the injury and spreads less rapidly, and presents in general a more varied anatomical and clinical picture than the first class of cases.

Hitschmann and Lindenthal consider that Pirogoff's first group corresponds especially to cases of pure or predominant infection with the gas bacillus, and his second group to mixed infections. Erdmann is also of the opinion that in the unmixed infections the emphysematous necrosis spreads more rapidly and is more likely to terminate fatally. On the other hand Muscatello and Gangitano, who also divide the cases into two groups — pure infections and mixed infections — hold that the mixed infections are characterized by the rapidly progressive form of emphysematous gangrene, while the pure infections, at least in their early stage, show little tendency to spread beyond the injured tissues. According to the last named authors the gas bacillus is incapable of exerting any pathogenic action upon healthy tissues, but attacks only tissues already altered in their vitality by injury, other pathogenic microörganisms, toxins, or other depressing factors.

The study of our cases has not enabled me to adopt either of the two conflicting opinions just stated. Of the

collected forty-six cases, thirty were mixed infections, fourteen were pure, and in two there is no clear statement on this point. The most common associated bacteria were the pyogenic streptococci and staphylococci. Among other forms found occasionally were Bacillus coli, proteus, pyocyaneus, tetanus, Sanfelice's Bacillus pseudo-œdematis maligni, and uncultivable, often spore-bearing, bacilli. It seems probable that the bacillus of malignant œdema, being a common inhabitant of the soil, must occasionally, like the tetanus bacillus, be present, but it was not isolated from any of these cases. My experience is that if reliance be not placed exclusively upon cultures, but careful microscopical examination be also made, instances of unmixed infection with the gas bacillus are rare. It is by no means always easy to determine whether associated bacteria are exerting pathogenic action or not. Pyogenic cocci may be present without producing pus or marked inflammatory reaction.

We have found rapidly progressive forms of emphysematous gangrene both with pure infections with the gas bacillus and with mixed infections, and on the other hand we have observed with both types of infection cases in which the tendency to spread is much less marked. In all of the cases with much purulent inflammation mixed infection was present, but aside from this feature the division of the cases into pure and mixed infections does not, according to our experience, correspond to different, sharply marked, clinical features. Pirogoff's classification is doubtless of clinical value, but there is every gradation between the two groups.

I cannot agree with Muscatello and Gangitano, whose investigations of emphysematous gangrene are of great value, that the gas bacillus is incapable of attacking healthy tissues. Virulent cultures, even in small doses, can produce rapidly spreading gaseous phlegmons when inoculated into the subcutaneous tissues of susceptible animals, such as guinea-pigs and pigeons. In human beings the emphy-

sema may extend very rapidly into the healthy tissues, frequently outstripping in its advance the inflammatory œdema. This may occur in pure, as well as in mixed, infections with the gas bacillus. It is true, as urged by Muscatello and Gangitano, that toxins derived from the bacilli may prepare the tissues for the invasion and action of the advancing bacilli, but the same explanation has been put forward for the spread of other pathogenic bacteria in healthy tissues. In asserting that the gas bacillus may attack healthy tissues, I would not be understood as minimizing the great importance of the various accessory causes of emphysematous gangrene which act by lowering the vitality of the tissues or the general resistance of the patient or as failing to recognize the marked resistance to infection by the gas bacillus offered by the healthy tissues, a resistance to which I have previously called attention and which I shall have occasion further to emphasize.

Gas may appear in the tissues as early as eight hours after the injury. In a case of gaseous phlegmon following a bullet wound of the knee joint, reported by Welch and Flexner, Dr. Bloodgood recognized gas in the joint and surrounding tissues just twenty hours after the injury.* In a case of Dr. Tiffany's, which we have reported, death from emphysematous gangrene, due to pure infection with the gas bacillus, occurred forty-eight hours after a fall causing a compound, comminuted fracture of the patella with grinding of the underclothing into the wound. There was no other injury of the body. Any one who has seen one of these rapidly fatal cases of spreading, traumatic, emphysematous gangrene will receive an impression which he will never forget.

The anatomical and clinical study of uncomplicated emphysematous gangrene demonstrates that the disease is not,

* It is to be regretted that in the cases reported by Prutz (Deutsche Zeitschr. f. Chirurg., 1898, xlviii., p. 591) as traumatic entrance of air into the knee joint, no bacteriological examination was made.

as many formerly supposed, simply an intense variety of ordinary phlegmonous inflammation or cellulitis, but is a disease sui generis. It may be combined with phlegmonous inflammation, but then some other microörganism, usually the streptococcus, is associated with the gas bacillus.

In typical uncomplicated cases the lesions consist in necrosis of all the tissues, the presence of gas in the interstices, infiltration with blood, evidences of the mechanical action of the gas, and exudation of a variable amount of bloody serum. The amount of gas varies much in different cases. There may be only a few bubbles or the tissues may be everywhere blown up with gas. The nuclei disappear by karyolysis. The notable thing in most cases is a nearly complete absence of leucocytes and of cellular reaction, although in a few instances, even of pure infection, I have found leucocytes in considerable number, and even purulent foci, but generally at a distance from the primary necrosis. As will be shown later Bacillus aërogenes capsulatus in certain situations is capable of setting up purulent inflammation. In one of our cases, reported by Bloodgood, Dr. Cushing found gas bacilli without gas in a small subcutaneous abscess of a stump two months after amputation of the thigh for emphysematous gangrene of the leg, the amputation having been followed by uninterrupted convalescence.

After death there may be rapid extension of the subcutaneous emphysema, and at autopsies made a few hours after death gas bubbles may be found in the heart, vessels, liver and other organs, but as to this occurrence there is no rule. As I have already explained wide-spread distribution of gas in the blood and organs in early autopsies indicates entrance of the bacilli into the circulation during life.

As is to be expected from the etiology of many of the cases of emphysematous gangrene, emboli of fat and of bone-marrow cells are common in the pulmonary capilla-

ries, but I do not know that in any case they contributed to the fatal result.

The prognosis of emphysematous gangrene is more favorable to-day than before the antiseptic period. The disease terminated fatally in 59 per cent. of the cases in my list. In the cases observed and treated at the Johns Hopkins Hospital—ten in number—the fatality was 50 per cent., but of these one was a gaseous phlegmon of the pelvis extending to the thigh, resulting from traumatic rupture of the rectum; a second was a gaseous phlegmon of the abdominal wall following removal of the appendix and complicated with diffuse gangrenous peritonitis, and a third case terminated twenty-five days after disappearance of a gaseous phlegmon of the thigh (treated by incisions) from a late streptococcus infection, gas and the gas bacillus having disappeared. Of the two remaining fatal cases, in one—emphysematous gangrene following compound fracture of the thigh—amputation was refused by the patient until the fourth day when he was in a state of collapse and died fifteen hours later, and in the other—compound fracture of the skull and both lower extremities—the patient died thirty hours after the accident, having never regained consciousness. When the disease is accessible to surgical treatment, is not complicated by other grave conditions, and is promptly recognized and treated, the prognosis, according to the experience of my surgical colleagues, Professor Halsted and Dr. Bloodgood, is not very unfavorable.

The clinical evidence seems to me to favor the view that, at least in most uncomplicated fatal cases, death is due to toxæmia. Norris was unable to demonstrate the presence of strong toxins in artificial cultures of Bacillus aërogenes capsulatus, but, as is well known, the same difficulty is encountered with many other pathogenic bacteria which are believed to produce poisons in the human body. Mus-

catello is also of the opinion that the constitutional symptoms are attributable to toxæmia.

The suggestion that death may be due to gaseous embolism is not new. Pirogoff and other of the older observers knew that gas bubbles may be found within the heart and vessels very soon after the death of patients from emphysematous gangrene. In the discussion on Langenbeck's paper on traumatic infiltration before the Society of German Military Surgeons in 1870, Senator* advocated the idea that death is caused by gas embolism. While the possibility of this occurrence as the cause of death may be admitted, there is at present no proof of this opinion. Gas-bubbles are by no means always found in the blood and organs after death from emphysematous gangrene, and the clinical histories of those in which they are found post mortem do not appear to differ from those where they are absent. In experimental gaseous phlegmons in guinea-pigs there is no evidence of the presence of gas bubbles in the circulation during life.

In one of our cases (Mann) death resulted from tetanus, and Verneuil† has reported three cases, which without bacteriological examination he calls malignant œdema, where tetanus intervened. There is nothing surprising in this association when one considers that the home of the tetanus bacillus, like that of the gas bacillus, is the soil. The period of incubation for tetanus is longer than for infection with the gas bacillus, the former appearing rarely before the seventh day, and the latter usually within two or three days after the injury.

Prophylactic measures against emphysematous gangrene are sufficiently obvious from the etiological factors which have already been considered. It is important in wounds of the character most frequently followed by this complica-

* Deutsche militärärztl. Zeitschr., 1872, I., p. 260.
† Semaine méd. 1890, p. 403.

tion to search microscopically and by cultures for the gas bacillus. The examination of cover-slips will usually suffice for a probable diagnosis. I have already cited instances in which this bacillus has been detected before the onset of emphysema and in which there is good reason to think prompt surgical treatment warded off severe infection. The possibility of infection from the intestinal canal, as well as from external sources, is to be borne in mind.

The cases were treated either by free incisions or by amputation or both. The results were better after amputation than after simple incisions. Of the cases of emphysematous gangrene affecting primarily the extremities, the recoveries numbered 68 per cent. after amputation, and $33\frac{1}{3}$ per cent. after incision without amputation. Careful study of individual cases shows that amputation is by no means always necessary. Everything depends upon early recognition of the nature of the infection. Dr. Bloodgood,* from a relatively large experience says: "If the infection is recognized early, and the destruction of the soft parts and bone is not extensive, free incisions, with immediate continuous bath treatment, should be tried. If the general symptoms of infection are not immediately relieved, amputation should be done. If, however, the infection is recognized late one should take no risk but amputate at once. . . An early diagnosis will probably save life, and from many observations an amputation may not always be necessary."

A similar position as to prognosis and treatment is taken by Muscatello and Gangitano in their valuable paper on gaseous gangrene, in which they also emphasize the value of abundant irrigations with disinfectant and oxidizing solutions. In the service of Dr. Halsted at the Johns Hopkins Hospital the continuous bath treatment has been found useful. Hitschmann and Lindenthal are certainly mistaken in their assertion that incisions are usually without favorable

* Op. Cit., p. 174.

effect, and that early amputations offer the only chance of recovery. As pointed out by Thorndike and others, recovery may follow amputation even when it is impossible to remove the whole of the diseased tissues.

### UTERINE INFECTIONS.

Knowledge of Bacillus aërogenes capsulatus is of not less interest and importance to the obstetrician than to the surgeon. Infection of the puerperal uterus by this microorganism leads to a variety of morbid conditions, some comparatively mild, others of the utmost gravity.

The wide distribution of the gas bacillus in the intestinal canal and the outer world renders as explicable the occasional presence of this bacillus in the female genital tract as that of the colon bacillus in the same situation. Lindenthal* found the gas bacillus in the vagina without the presence of gas or other pathogenic effects twice out of six puerperal cases examined. According to the observations of others, Lindenthal's experience would seem to be exceptional. As with so many other pathogenic bacteria the mere presence of the organism upon exposed surfaces does not necessarily signify infection.

I shall consider the uterine infections by the gas bacillus under the headings of emphysema of the fœtus, puerperal endometritis, physometra, emphysema of the uterine wall, and puerperal gas-sepsis, although these conditions may be associated with each other.

*Emphysema of the Fœtus.*—The occurrence of emphysema of the dead fœtus in utero has been known for centuries,† but it was not until 1897, when Dobbin published his paper on "Puerperal Sepsis due to Infection with the

* Wiener klin. Wochenschr., 1897, pp. 3 and 35.

† It is usually stated that this condition was known to Celsus, but he does not expressly mention the presence of gas, although this is to be inferred. In the chapter on the extraction of the dead fœtus, he says: "Solet etiam evenire, ut is infans humore distendatur, exque eo profluat foedi odoris sanies." Milligan's "Celsus," p. 394, Edinburg, 1831.

Bacillus aërogenes capsulatus"* that the cause of this condition was determined to be invasion by the gas bacillus. In this case gas and the gas bacillus were both recognized during the life of the patient in the fœtus, the placenta and the cavity of the uterus, and after death there was general gas formation throughout the body.

Dr. Dobbin has kindly given me the notes of a second, unpublished case observed by him. He was called to see a woman in difficult labor, upon whom repeated attempts at delivery of a dead fœtus had been made. Upon examination he recognized crepitation of the caput succedaneum, while the fœtus was still in utero. Upon delivery the fœtus was emphysematous with foamy organs. The gas bacillus was found in pure culture in the fœtus and mixed with other bacteria in the amniotic fluid. No gas was recognized in the uterus after delivery. The patient made a good recovery without evidence of sepsis.

Menge and Krönig† have observed three instances of fœtal infection with the gas bacillus, and have brought strong evidence that usually the invasion is through the mouth of the fœtus, the bacilli being taken into the lungs or stomach by inspiring or swallowing amniotic fluid. In their cases the bacilli were not found in the part of the umbilical cord attached to the placenta, but in Dobbin's first case this as well as the placenta itself was infected with the gas bacillus. The amniotic fluid within the intact membranes, as is well known, is usually sterile, and only exceptionally becomes infected before rupture of the membranes, so that the infection of the fœtus from this source is generally after this rupture. As shown by Menge and Krönig all grades of invasion of the fœtus by the gas bacillus occur, so that there may be only a small amount of gas, and this limited to the lungs or the alimentary canal or both. The medico-

* Bulletin of The Johns Hopkins Hospital, 1897, VIII., p. 24.
† Bakteriologie des weibl. Genitalkanales, Teil II., p. 167, Leipzig, 1897.

legal importance of not mistaking for air this gas in the fœtal lungs produced by gas bacilli should be emphasized.

As is well known, fœtal emphysema is usually unattended with danger to the mother. In two of the five cases due to Bacillus aërogenes capsulatus in my list the puerperium was even without rise of temperature, in two there was mild fever, but in Dobbin's first case there was rapidly fatal gas sepsis.

*Puerperal Endometritis.*—Under the heading "gas-sepsis" will be considered cases of acute uterine infection with the gas bacillus, followed by invasion of the blood and organs by this organism. Here it may be mentioned that Bacillus aërogenes capsulatus may be present in the uterus, usually in association with other bacteria, in both mild and severe cases of puerperal endometritis without the recognition of gas in the fœtus, or the uterine cavity, or wall. An example of such a case is an unpublished one in the service of Dr. J. Whitridge Williams, of which the notes have been furnished me by Dr. Dobbin. A woman with rhachitic pelvis, upon whom delivery by forceps had been attempted before admission to the Johns Hopkins Hospital, was there delivered by craniotomy. There was no gas in the fœtus. Forty-eight hours later Bacillus aërogenes capsulatus, together with streptococci, was found in the uterine lochia. No gas was detected. The patient developed fever, but recovered.

In two cases, one reported by Dobbin* and the other by Blumer,† the gas bacillus, although not identified with absolute certainty, was probably present in the puerperal uterus in association with the typhoid bacillus.

*Physometra.*—Distension of the uterine cavity with gas (physometra or tympany of the uterus) was present in Dobbin's first case, already cited, and is often associated

* Am. Journ. Obstetrics, 1898, XXXVIII, p. 185.
† Ibid. 1899, XXXIX., p. 42.

with emphysema of the dead fœtus, but may occur without the latter, and even in the non-pregnant uterus. This curious condition was formerly ascribed to entrance of air or to ordinary putrefaction,* but it is now known to be the result of the activity of gas-producing bacilli.

Since the observations of Lindenthal,† reported in 1898, it cannot be doubted that Bacillus aërogenes capsulatus is the chief cause of physometra. He found this bacillus in five cases during life, and reproduced the condition experimentally in guinea-pigs. He is justly skeptical of the correctness of the previous reports of Gebhard‡ concerning the agency of the colon bacillus in generating gas within the uterus. We know from Theobald Smith's investigations that the colon bacillus can produce gas only from carbohydrates, whereas it is the most distinctive biological attribute of our gas bacillus that it can produce gas from proteids. Until it has been shown that the amniotic fluid and uterine contents may, under any conditions, contain enough carbohydrate to explain the development of gas by the colon bacillus, there is every reason to question the claims for this bacillus as a cause of tympany of the uterus.§

My list of cases contains ten instances of physometra in which Bacillus aërogenes capsulatus was demonstrated. Although, as some of our cases show, this condition may be associated with invasion of the bacilli into the wall of the uterus, and by acute gas sepsis, these occurrences are exceptional, and the prognosis is in general a favorable one. Most of the cases furnish a good illustration of the resistance of living human tissues to the action of the gas bacil-

* The older hypotheses and records on physometra and emphysema of the fœtus are fully presented by Staude, Zeitschr. f. Geb. u. Gynäk., 1878, III., p. 191.

† Monatsschr. f. Geb. u. Gynäk., 1898, VII., p. 269.

‡ Zeitschr. f. Geb. u. Gynäk., 1893, XXVI., p. 480, and 1897, XXXVII., p. 132.

§ Halban (Monatsschr. f. Geb. u. Gynäk., 1900, XI., p. 102), states that he has found lactose once in the amniotic fluid of a normal puerpera. Further studies of this subject are needed.

lus. Doubtless, in these cases, the bacilli grow simply in the amniotic fluid after rupture of the membranes, and in the dead fœtus, these offering no vital resistance, whereas we must suppose that the intact uterine wall offers ordinarily an effective resistance to the invasion and multiplication of the gas bacillus. That occasionally the bacilli may find other dead material in the uterus, as in sloughing myomata and cancers, is evident from the histories of some cases of physometra.

*Emphysema of the Uterine Wall.*—Far graver in significance is septic emphysema of the uterine wall of which Halban* has recently reported an interesting case due to Bacillus aërogenes capsulatus. Graham, Steward and Baldwin and P. Ernst were the first to demonstrate this bacillus in this condition, their papers, to which reference has already been made, appearing simultaneously in August, 1893. Eleven instances of emphysema of the wall of the uterus, all puerperal, have been reported, of which five were recognized during life. In all but Halban's case there was also physometra, and this would seem to be a necessary accompaniment unless the cervical canal is open, so as to permit the escape of the gas from the uterine cavity. All of the cases were fatal, and in most gas was found at autopsy in the blood and internal organs.

Subperitoneal emphysema is a condition which has been observed after rupture of the uterus, Dischler† having collected reports of 14 cases. In most instances this has been attributed to entrance of air, but I think that it is safe to predict that the gas bacillus will be found in similar cases in the future, if proper methods for its detection are employed. I know, however, of no instance of this condition in which it has been looked for.

*Puerperal Gas Sepsis.*—I have adopted from Halban the term "gas sepsis" as a convenient one, although per-

* Monatsschr. f. Geb. u. Gynäk., 1900, XI., p. 88.
† Arch. f. Gynäk., 1898, LVI, p. 199.

haps open to criticism, to designate the important group of fatal puerperal cases in which gas bubbles are found at early autopsies in the heart and vessels and often also in the organs and tissues under conditions where we must suppose that gas bacilli and possibly gas have passed from the uterus into the circulation during life.* Here in my opinion belong most of the cases which have been reported as deaths due to the entrance of air into the uterine veins. This opinion, which I expressed in my first communications on the gas bacillus in 1891 and 1892, received prompt confirmation in the papers of Graham, Steward and Baldwin and P. Ernst in August 1893, and has since been strengthened by other similar observations. Cases described by Wendeler† as sepsis acutissima belong also in the same category.

My list includes twelve puerperal cases in which gas bubbles and the gas bacillus were found at autopsy in the blood or organs, but in only six of these does it seem to me conclusive or extremely probable that the infection occurred during life.‡

The most remarkable of these cases is that reported by Graham, Steward and Baldwin of a woman, upon whom abortion had been recently produced, who during the four hours immediately preceding death became emphysematous over nearly the whole body. At the autopsy gas and gas bacilli were found everywhere throughout the body. In Dalton and Bremer's§ case, also one of criminal abortion, an emphysematous swelling of the arm and pectoral region was likewise recognized during life. These cases are of import-

* Gas sepsis in this sense occurs also in other than uterine infections with the gas bacillus.

† Monatsschr. f. Geb. u. Gynäk., 1896, IV., p. 581.

‡ These are the cases reported by Graham, Steward and Baldwin, P. Ernst, Krönig and Menge, Dobbin, F. C. Wood, (Med. Record, Apr. 15, 1899), and Halban, to whose papers references have already been given.

§ Am. Journ. Med. Sc. 1888, XCV., p. 594. This infection, although attributed to the bacillus of malignant œdema, was probably due to the gas bacillus.

ance as demonstrating the invasion of the body by the gas bacillus from the uterus while the blood is still circulating. In Halban's and Dobbin's cases gas was also recognized during life within the wall or cavity of the uterus.

In the majority of cases of puerperal gas sepsis there has been some operative interference preceding infection, such as criminal abortion, forced delivery for placenta prævia or other causes, or the manipulations of an unskilled midwife. The fulminating character of the infection, death being sometimes very sudden, is a notable feature of many of the cases.

In a case of attempted criminal abortion reported by Perkins* the patient, according to the statement of the physician in whose office death occurred, died suddenly, and at the autopsy, twelve hours later, in cold weather, gas was found in the vena cava, heart, and other vessels, with evidences of injury to the pregnant uterus. The case was reported by Perkins as one of death from air embolism, and certainly with as much and even more plausibility than most cases thus reported. After the publication, Dr. Perkins, upon the request of Dr. Dobbin, was so good as to send the uterus, well preserved in alcohol, to my laboratory where Dr. Dobbin demonstrated in the uterine vessels and tissue bacilli morphologically and in staining reaction identical with Bacillus aërogenes capsulatus.†

I would not be understood to deny the possibility of the occurrence of fatal air embolism from the uterus. A very few of the reported cases are difficult to interpret upon any other supposition, but I do claim that the foundations of this doctrine have been seriously shaken by our discovery and investigations of the gas bacillus, and that no case, however plausible, can be considered as positively proven without a satisfactory bacteriological examination. The limitation of

* Boston Med. and Surg. Jour., 1897, CXXXVI., p. 154.

† Dobbin has reported his examination with further notes of this case in the Monatsschr. f. Geb. u. Gynäk., 1897, VI., p. 375.

gas to the right heart and adjacent vessels may occur from invasion by the gas bacillus and is not, as is often represented, peculiar to air embolism. Did we not know how long it takes new knowledge, especially that originating in this country, to penetrate throughout the medical world, it would be amazing that cases should still continue to be reported, as they are,* of deaths ascribed to air embolism without any bacteriological examination or even any reference to the possibility of any other explanation. I am not aware that in any instance of alleged air embolism, a bacteriological examination has been made, which would exclude the presence of gas-forming bacteria.

Whether, as suggested by Staude, in any of the cases with gas within the uterine cavity, death is attributable to gaseous embolism, is, I think, an open question. It is possible, although I know of no proof of it, that in some of the cases of sudden death during or immediately after some manipulation or operation on the pregnant uterus and attributed to air embolism, gas, generated by bacteria, may have existed under pressure within the uterine cavity and have entered wounded veins in sufficient amount and so suddenly as to have caused death.

To what extent the free gas found in the blood-vessels, heart and internal organs, even very soon after death from what has been described as puerperal gas sepsis, is there during life, is a question difficult to answer. I have already considered this subject, and in this connection shall again emphasize the importance of caution in interpreting the presence of gas in these situations as a vital phenomenon, although there is evidence that it may be such.

### INFECTIONS OF THE URINARY TRACT.

There is evidence that the urinary tract may not only be a portal of entrance for the gas bacillus into the circulation

* Zorn (Münch. med. Woch., 1898, p. 567) may be cited as an example.

or adjacent tissues but also be itself the seat of infection by this organism. Unfortunately for the decisive interpretation of many of these cases, Bacillus aërogenes capsulatus has, so far as I am aware, hitherto been found in the urinary tract only after death, although in some instances so soon thereafter and under such conditions that its presence during life cannot be doubted.

I have already called attention to instances of emphysematous gangrene following external urethrotomy and other operations on the urinary passages.

Among the cases of general invasion of the blood and organs (foamy organs) by the gas bacillus observed by myself and others are several in which the portal of entry was the urethra, bladder or other part of the urinary tract.* In a case of urethral stricture with cystitis, for which perineal section had been done, reported by Welch and Flexner, gas bacilli were found *three quarters of an hour after death*, in large numbers in the bladder, ureters and renal pelvis and a few gas bubbles and gas bacilli were already present in the blood of the right ventricle. In Howard's case of meningitis, caused by the gas bacillus, to be cited subsequently, he considers that the portal of entry was the urinary tract.

Gas, generated by Bacillus aërogenes capsulatus, has been found in the urinary passages in six cases which have come to my notice.† The gas may be either free in the cavity of the bladder, ureters or renal pelvis, or contained within submucous blebs, or in both situations.

Welch and Flexner‡ have reported an instance of pneumaturia in a diabetic man in whom six hours after death, without trace of cadaveric decomposition, the urinary bladder was found filled with frothy urine containing Bacillus

* Such cases are reported by Welch and Flexner, Goebel, Dunham, and Howard in papers already cited.

† These do not include some instances of very extensive post-mortem emphysema of the organs with gas everywhere throughout the body.

‡ Case XIII of our list in Journal of Experimental Medicine, 1896.

aërogenes capsulatus in pure culture. This case indicates that the colon bacillus and Bacillus lactis aërogenes are not the sole causes of pneumaturia in diabetics. Dr. Flexner has given me the notes of a second case of pneumaturia upon which he made the autopsy at the University Hospital, Philadelphia. This was of a patient with chronic cardiac disease, who had been catheterized twenty-eight hours before admission, and who died thirty hours after admission. At the autopsy, made one hour and a half after death, about 60 cc. of frothy urine were found in the bladder, from which Bacillus aërogenes capsulatus, together with Staphylococcus aureus and Streptococcus pyogenes (no colon bacilli), was cultivated. Neither gas nor gas bacilli were found elsewhere in the body. The mucous membrane of the bladder was œdematous. There can be little doubt that in this case the gas bacillus was introduced by the catheter into the bladder.

In one of Welch and Flexner's cases (Case XVIII) of hypertrophied prostate with pyoureter and pyonephrosis the renal pelves and ureters were found at autopsy distended with gas and containing pus mixed with bubbles of gas. Small gas cysts were present in the mucous membrane of the renal pelves. Neither gas nor gas bacilli were present outside of the urinary organs. In this case cocci and colon bacilli were associated with the gas bacillus.

In a case of Dr. Kelly's, of which the full records have been given me by Dr. Miller,* there was pneumaturia demonstrated by cystoscopic examination and ureteral catheterization to come exclusively from the inflamed left renal pelvis and ureter. Among cocci and other bacteria were found on cover-slips bacilli morphologically resembling Bacillus aërogenes capsulatus, but unfortunately no anaërobic cultures were made. None of the bacteria which grew

* This case is briefly reported by Kelly and MacCallum, Journ. Amer. Med. Assoc., 1898, XXXI., p. 376, whose paper may be consulted for the full literature of pneumaturia.

aërobically produced gas in lactose agar. This case is interesting as demonstrating that the gas may come exclusively from one renal pelvis and ureter, but the microörganism producing the gas was not satisfactorily demonstrated. It seems certain that it was an anaërobic organism, and from the microscopical appearances may have been the gas bacillus.

The subject of submucous gas cysts will be considered subsequently; but here it may be mentioned that besides the gas cysts in the renal pelves noted by Welch and Flexner in the case already cited, Goebel found gas blebs, containing in pure culture Bacillus aërogenes capsulatus, beneath the mucous membrane of the urinary bladder, without gas elsewhere in the body, and Dunham has reported a like condition of the bladder in a case of emphysematous gangrene with general invasion of the blood and organs by the gas bacillus.

Bacteria have been found also in emphysema of the bladder (cystitis emphysematosa) by Eisenlohr,* Camargo† and Kedrowsky‡; but it is impossible, from the authors' descriptions, to identify their bacteria. Kedrowsky considers the bacillus isolated by him as allied to Bacillus aërogenes capsulatus, but less sensitive to oxygen. Perhaps it was Sanfelice's Bacillus pseudo-œdematis maligni, to which I have already referred, but Kedrowsky's description of his cultures hardly inspires confidence in their purity.

Welch and Flexner, and Howard have reported finding the gas bacillus, in association with other bacteria, in inflammatory lesions of the bladder, renal pelvis and kidneys, without the detection of gas. Howard considers that in one of his cases the bacillus was concerned in the etiology of suppurative lesions of the kidneys, but in this case the colon bacillus and Streptococcus pyogenes were also present.

* Ziegler's Beitr., 1888, III., p. 101.
† Thèse de Genève, 1891.
‡ Centralbl. f. allg. Path. u. path. Anat., 1898, IX., p. 817.

## INFECTIONS DERIVED FROM THE GASTRO-INTESTINAL CANAL.

Mention has already been made of the frequent, if not constant, presence of Bacillus aërogenes capsulatus in the intestinal canal, of gaseous phlegmons originating from this source, and of the readiness with which intestinal bacteria may gain access to the genito-urinary tract.

The intestine is by far the most common source of the gas bacilli found together with gas bubbles in the blood and organs at autopsies. This invasion may occur either with or without definite intestinal lesions, and is probably in the majority of cases an agonal or post mortem event. The mode of distribution and spread of the bacilli in these cases has been well described by Howard,* whose experience has been exceptionally large with this class of affections. Especially demonstrative of invasion of gas bacilli from the intestine, usually post mortem, is the occurrence of gas bubbles limited to the neighborhood of the intestine, as in the intestinal wall, within the portal or mesenteric veins or lymphatics, in the subperitoneal tissue, mesentery and omenta, around the pancreas, in the mesenteric glands, and especially in the loose tissue near the gall-bladder and in the porta of the liver, without gas in more remote situations. I have seen examples of each of these occurrences in very early autopsies without ordinary cadaveric decomposition.

*Local Gastro-Intestinal Lesions.*—Interstitial emphysema of the gastro-intestinal wall will be considered subsequently.

Howard has described several cases with larger or smaller areas of superficial necrosis of the mucous membrane of the stomach and intestine, in which gas bacilli were present in large numbers. These areas, which may occur either with

* Op. cit. p. 491.

or without gas cysts, are found most frequently beneath the folds of the valvulæ conniventes and are characterized by absence of nuclear staining and disintegration of the cells and tissue, usually without marked inflammatory reaction.

Bacillus aërogenes capsulatus may undoubtedly be a cause of meteorism. Instances of this are reported by Welch and Flexner and by Howard.

*Pneumo-peritonitis with and without Perforation.*—My records include thirteen cases of diffuse peritonitis in the exudate of which Bacillus aërogenes capsulatus was found. Eleven of these were in autopsies made either by Dr. Flexner or myself, seven having already been published by us in 1896. The remaining two cases (both being perforations of gastric ulcers) have been reported by Page,* and by Pratt and Fulton.† It was the observation of these cases which first called my attention to the frequent presence of the gas bacillus in the intestine.

Ten of the cases were perforative and three were non-perforative. Of the former four were the result of perforation of typhoid ulcers, four of gastric ulcers,‡ one of strangulated, gangrenous intestine and one of a cancerous ulcer of the duodenum. In the last case (autopsy fourteen hours after death) the exudate was sero-fibrinous and the gas bacillus was found in pure culture,§ and abundantly in the peritoneal cavity and was absent from other organs and the blood. In the other perforative cases the gas bacillus was mixed with other bacteria, although in some instances it predominated. In all of the cases the abdomen was

* Canada Lancet, May, 1900.

† Boston Med. and Surg. Journ., June 7, 1900, p. 599.

‡ Welch and Flexner have reported an instance of peritonitis from perforated gastric ulcer in a rabbit, in which the gas bacillus was found.

§ The purity of the culture in this case is explicable by the results of Cushing and Livingood's interesting bacteriological and experimental studies of the duodenal flora published in Contributions to the Science of Medicine, dedicated by his pupils to William Henry Welch on the 25th Anniversary of his Doctorate, p. 543, Baltimore, 1900.

greatly distended with gas and usually there was great tympanites.

I attach especial importance to the case fully reported by Welch and Flexner,* in which we brought conclusive evidence of the occurrence of pneumo-peritonitis without perforation, the first of the kind on record in which similar proof was obtained. Since our publication a similar case has been observed by Dr. Flexner in Manila, who has kindly furnished me the notes. At the autopsy, twelve hours after death, there was found hemorrhagic infarction of the lower part of the ileum and adjacent part of the large intestine, caused by the passage of this part of the intestine through a hole in the mesentery. The peritoneal cavity was greatly distended with gas which burnt with a pale, blue flame. There was a large amount of frothy, bloody serum in the peritoneal cavity together with a fibrinous exudate. The most careful examination showed no perforation. Bacillus aërogenes capsulatus was obtained in pure culture and abundantly from the peritoneal fluid. Gas was absent from the blood and other organs.

We have found the gas bacillus, mixed with other bacteria, twice in circumscribed, gas-containing, intraperitoneal abscesses resulting from perforation of the appendix vermiformis.

*Hepatic and Biliary Infections.*—The development of gas in the liver is so striking a phenomenon in most autopsies where the gas bacillus and free gas are found in the blood and organs that P. Ernst used the term "Schaumleber" for the title of his article on the gas bacillus, published a year after the paper by Welch and Nuttall, in which we first directed attention to the subject of foamy organs and the gas bacillus.† I have already considered

* Op. cit. p. 36.

† Heydenreich's paper on "Emphysem der Leber" (Centralbl. f. Bakter., 1897, XXI., p., 305) may be mentioned as a curiosity. He had never heard of the gas bacillus or of any other investigation of the subject later

the general subject of gas bubbles in the blood and organs, and wish here to call attention especially to infections of the gall bladder and biliary passages by Bacillus aërogenes capsulatus.

In cases of foamy liver gas may be found in the bile ducts and gall bladder, but my experience is that, when the gas bacilli reach the liver through the blood vessels, the appearance of gas in these situations is a rather late occurrence and met chiefly in advanced cases. In contrast to these cases are the observations of gas in the biliary passages, associated sometimes with definite lesions of the bile ducts and liver, where the evidence is that the gas bacilli entered from the intestine directly into these passages. Two such cases have been reported by Howard,* and I have observed two cases.

Pratt and Fulton † report a remarkable case of cancer of the common bile-duct and pancreas in which cholecystotomy was performed, the opening in the gall bladder being stitched to the abdominal walls. At the autopsy the liver was found studded with minute abscesses with greenish translucent walls. In cover slips, sections and cultures Bacillus aërogenes capsulatus was found in pure culture in these small biliary abscesses, but there was no gas in the abscesses, the liver, blood, or other organs. A somewhat similar case, in which cholecystenterostomy for gall stones had been performed, was previously reported by Nicholls‡ from Adami's laboratory. Here also multiple miliary abscesses containing the gas bacillus were found in the liver, but in this case gas was present in the liver, as well as in the blood and other organs, the autopsy being six hours

than 1872. In contrast to this is the interesting article of P. Bernhardt on pneumathemia and foamy organs with full consideration of the recent literature. Deutsche med. Woch., 1900, p. 83.

* Op. cit. pp. 475 and 476.

† Boston Med. and Surg. Journ., June 7, 1900, p. 599.

‡ Brit. Med. Journ., 1897, II., p. 1844.

after death. Larkin* has likewise reported a case of hemorrhagic pancreatitis with fat necroses and small, multiple hepatic abscesses with gas holes in the liver, from which Bacillus aërogenes capsulatus was isolated. The autopsy was eight hours after death.

Hintze† has recorded a post-mortem observation of gas in the inflamed bile ducts with cholelithiasis. He cultivated only the colon bacillus, but it does not appear that he made anaërobic cultures.

From the foregoing cases it is to be inferred that the gas bacillus may invade the bile ducts and gall bladder from the intestine, and sometimes during life, and that it may not only produce gas but also necroses and purulent inflammation. The presence of gall stones, cancer of the bile ducts and operations on the gall bladder appear to favor this mode of infection.

Rist‡ found the gas bacillus (Bacillus perfringens), without gas or other lesions attributed to it, in an extirpated gall bladder containing a calculus and clear viscid fluid.

### INTERSTITIAL EMPHYSEMA OF THE GASTRO-INTESTINAL, GENITO-URINARY AND BILIARY TRACTS.

One of the most interesting lesions produced by Bacillus aërogenes capsulatus is the formation of submucous or subserous gas cysts or blebs, of which the earliest examples attributed to this organism were reported by P. Ernst, Goebel, and Welch and Flexner. They are sufficiently common to have been observed by nearly all investigators who have had much experience with the gas bacillus in human beings. My list of cases includes twenty-five instances of this condition, of which five were of the stomach, eleven of the intestine, far oftener of the small than of the

* Med. Record, 1898, LIII., p. 354.
† Münch. med. Woch., 1895, XLIII., p. 209.
‡ Thèse, Paris, 1898.

large intestine, five of the gall bladder and bile ducts, three of the urinary bladder, one of the renal pelvis, and one of the vagina. The majority of the cases were observed by Flexner, Howard, and myself, and in all the gas bacillus was demonstrated. The case of emphysema of the vagina was reported by Lindenthal.*

These gas cysts vary in size from microscopic dimensions to large blebs. They are most common in the submucous coat, but may be present in the mucous membrane, the muscular coat or beneath the serous covering; in fact in any part of the membranous wall. They may be few or in enormous numbers, in groups or scattered. In one of our cases the whole small intestine from the duodenum to the ileo-cecal valve was studded with small gas cysts. Gas cysts of the same general character may be found in the mesentery and omenta.

These gas cysts are, in their inception, simply such gas holes as we are already familiar with in the liver and other organs in cases of local or general invasion with the gas bacillus. They indicate a foamy or emphysematous condition of the walls of the stomach, intestine, gall bladder, bile ducts, urinary bladder and vagina, due to the invasion into these parts of the gas bacillus.

The condition of the walls of the blebs and of the surrounding tissues varies. Some of the cavities are round and their walls smooth, others are irregular in shape and have ragged walls. They may correspond to dilated lymphatics, but more frequently they do not represent dilatation of any preformed channels. There may be communication between adjacent cavities, but oftener the cysts are distinct from each other. The tissue in the immediate neighborhood of the cavities may present no alteration not explicable by the mechanical pressure of the gas, or it may show necrosis in varying degree and extent. Inflammatory

* Wiener klin. Woch., 1897, pp. 3 and 35.

changes or cellular reaction, which could be reasonably referred to the presence of the cysts or of the gas bacilli causing them, were not noted. Sometimes a little coagulated, homogeneous or granular material is present in sections of the cysts in hardened tissues, as indeed may often be found in gas holes in the organs.

In sections stained by Gram there is usually no difficulty in demonstrating the relation of the gas bacilli to the cysts. The bacilli may be in masses in the walls of the cysts, but sometimes they are not more numerous there than in the tissue at a distance from the gas blebs, and occasionally it requires some searching to find them. By anaërobic methods the gas bacillus can be cultivated from the cysts or the adjacent tissue.

All of the instances of submucous and subserous gas cysts in my list were observed post mortem. In the larger number of cases gas bacilli and gas bubbles were more or less widely distributed in the blood and organs, but without evidences of ordinary post mortem decomposition. There are, however, several cases in which these gas cysts, in various situations, were the sole manifestation of the presence of the gas bacillus in the body. Howard has shown that if careful, systematic search is made for areas of necrosis and minute gas cysts due to Bacillus aërogenes capsulatus in the stomach and intestine, these lesions can be found much more frequently than has been supposed.

It is, with our present information, a difficult matter to say in how many of these cases the emphysematous state existed before death. It is certain that, at least in the great majority of cases in my list, the emphysema was not the result of ordinary post mortem decomposition. In a large number of the cases the autopsy was made within a few hours after death. Howard, in one of his cases of gas cysts, limited to the intestine, made the autopsy one hour after death. I have already emphasized the importance of

great caution in interpreting as vital processes the various gaseous conditions of parts and organs not open to inspection during life, even when autopsies are made soon after death and there is entire absence of putrefaction, and I can only repeat this caution here. Mere absence of nuclear staining around gas holes, I do not regard as proof of their origin during life.

It would lead altogether too far to enter here into a discussion of the general subject of submucous gas cysts, concerning which there is a large literature, the principal references to which will be found in the articles of Eisenlohr,* Camargo,† Winands,‡ Dupraz§ and Lindenthal.‖ It would appear from a study of the records of the subject that anatomically different conditions have been described under the designation "gas cysts" (cystides aëriferæ). Some have been apparently of long standing and show chronic inflammatory changes in their walls and surrounding tissues. The most voluminous records relate to the gas cysts of the vagina (kolpohyperplasia cystica of Winckel, emphysema vaginæ of Eppinger).

At present we have no warrant to identify the more chronic gas cysts with thickened walls with the more acute emphysematous condition which I have described as referable to Bacillus aërogenes capsulatus. Nevertheless, the former are probably of bacterial origin also, although I am unable to accept the bacteriological findings of Eisenlohr, Camargo and Dupraz as at all convincing; for they made no use of anaërobic methods of cultivation. It is quite possible that Bacillus aërogenes capsulatus is concerned also in the etiology of the gas cysts of long standing; certainly it is important to apply hereafter anaërobic cultural methods to

* Ziegler's Beiträge, 1888, III., p. 101.
† Thèse de Genève, 1891.
‡ Ziegler's Beiträge, 1895, XVII., p. 38.
§ Arch. de méd. expér., 1897, IX., p. 282.
‖ Wiener klin. Wochenschr., 1897, pp. 3 and 35.

their study. Lindenthal has no hesitation in identifying the emphysema of the vagina observed by him post mortem, and from which, in a single case, he cultivated what he chooses to call Bacillus emphysematis vaginæ (in reality our Bacillus aërogenes capsulatus), with Winckel's kolpohyperplasia cystica; but, while he may be correct, further observations are needed before this anatomical and etiological identification can be accepted.

The only instance in which gas cysts of the human intestine have been recognized during the life of the patient is reported by Hahn,* who found them in large number upon opening the abdomen. It is to be regretted that no thorough bacteriological examination of this case was made.

In my original communication on the gas bacillus I ventured the surmise that the bacillus found by E. Fraenkel† in hardened sections from a case of gastritis emphysematosa might be identical with Bacillus aërogenes capsulatus, and this suspicion has gained in probability by Goebel's statements, based upon an examination of the sections, his work having been done under Fraenkel's supervision.

Notwithstanding an effort at compression, so much space has been occcupied in the presentation of the foregoing subdivisions of my subject, which in less than a decade has grown to considerable magnitude, that I shall only summarize very briefly a few remaining topics, although all deserve fuller treatment.

*Pulmonary and Pleural Infections.*—To the two instances of invasion of the lungs by the gas bacillus, reported by Welch and Flexner, I can add the cultivation of this organism from a gangrenous lung by Dr. Flexner in my laboratory. It has been found also by Guillemot‡ and Rist§ in pulmonary gangrene.

* Deutsche med. Woch., 1899, p. 657.
† Virchow's Archiv., 1889, CXVIII., p. 526.
‡ Thèse, Paris, 1899.
§ Thèse, Paris, 1898.

Of much importance is Levy's* demonstration of Bacillus aërogenes capsulatus as a cause of pneumothorax without perforation. This case, and those of pneumo peritonitis without perforation, to which I have already referred, have settled affirmatively the long standing controversy concerning the possibility of the generation of gas within closed serous sacs during life. Nicholls† has reported an instance of pneumothorax and pneumopericardium without perforation, in which the gas bacillus was probably present. Rendu and Rist‡ also isolated the gas bacillus (Bacillus perfringens) in a case of putrid pleurisy with gas. May and Gebhart§ and Finley‖ attribute their two cases of pneumothorax without perforation to the colon bacillus; but in neither were anaërobic cultures made. It is important that in all gaseous affections anaërobes should be searched for.

*Gas Bacillus in the Blood during Life.*—Extremely interesting is the demonstration by Gwyn,¶ both by coverslip specimens and by cultures, on repeated examinations, of Bacillus aërogenes capsulatus during life in the blood of a patient, in the Johns Hopkins Hospital, with chorea insaniens and acute endocarditis. I had the opportunity of examining his cultures, which were entirely typical. The patient died, but unfortunately no autopsy could be obtained. There was no evidence during life of free gas in the blood or tissues. The case is of importance as demonstrating that gas bacilli may be in the circulating blood for days in sufficient number to be demonstrable, both microscopically and culturally, without evidence of freegas. I have already expressed my suspicion that Achalme's anaërobic bacillus, which has likewise been cultivated from the

* Arch. f. exp. Path. u. Pharm., 1895, XXXV., p. 335.
† loc. cit.
‡ Bull. et mém. Soc. méd. d. hôp. de Paris, 1899, 3. s., XVI., p. 133.
§ Deutsches Arch. f. klin. med., 1898, LXI., p. 323.
‖ Philadelphia Monthly Med. Journ., 1899, I, p. 569.
¶ Bulletin of The Johns Hopkins Hospital, 1899, X., p. 134.

blood, as well as from the tissues, of several cases of acute articular rheumatism, is identical with Bacillus aërogenes capsulatus.

*Presence of the Gas Bacillus without Gas.*—In this connection I may say that the gas bacillus may be present and even multiplying within the human body without the production of gas. Certain organs, above all the liver, offer much more favorable pabulum for the generation of gas than do others; but even in the liver the bacilli may be present without gas. I have found gas bacilli in small clumps within the spleen and kidney, surrounded by areas of necrosis, without recognizable gas. I have already cited the demonstrations by Cushing and by Pratt and Fulton of the gas bacillus in small abscesses without gas, the observation of the latter being particularly complete and satisfactory. Rist* has cultivated the gas bacillus (Bacillus perfringens) from cases of fetid otorrhœa and of mastoid abscess. He does not mention the presence of gas in these cases.

Dr. Harris, in my laboratory, isolated the gas bacillus in pure culture from an abscess containing blood and pus in the neck of a dog, following an operation on the jugular vein. There was no gas in the abscess. Aërobic cultures were entirely negative. These observations, however, are not the only ones demonstrating that the gas bacillus may, under certain circumstances and in certain situations, manifest pyogenic capacity.

*Meningitis. Pyogenic Capacity of Gas Bacillus.*—Howard† in April, 1899, reported a case of acute fibrinopurulent meningitis, following operation for urethro-perineal fistula. Bacillus aërogenes capsulatus was found in pure culture in the meningeal exudate. Hitschmann and Lindenthal‡ have recorded another convincing example of acute

* Thèse, Paris, 1898.
† Bulletin of The Johns Hopkins Hospital, 1899, X., p. 66.
‡ Op. cit.

cerebral meningitis, following fracture of the occipital bone, in which the gas bacillus was found unmixed with other bacteria. These authors call attention to the pyogenic power of the gas bacillus when it attacks the meninges, but, as already shown, the same power may be manifested elsewhere.

*Cavities in the Brain.*—Finally I would call attention to Reuling and Herring's* and Howard's studies of cavities in the brain produced by Bacillus aërogenes capsulatus and to the light which their observations shed upon certain obscure examples of cerebral cavities previously reported.

In this excursion into pneumato-pathology I have invited you to the survey of fields comparatively new and little trodden. I can only hope that our journey has been not without some interest and some profit to you. To me the opportunity to present before such a body as The Massachusetts Medical Society the results of these investigations is one which I highly appreciate.

I desire in closing to pay my tribute of respect and to call to your grateful remembrance the founder of this lectureship, Dr. George Cheyne Shattuck. That the subject of this address would not have been without interest to him may be inferred from the title of his first Boylston Prize dissertation on the theme propounded in 1806 and entitled: "The difference between mortification produced by an external cause and that which is produced by a constitutional defect, the diagnosticks and proper mode of treatment of each.†" By his noble character, professional services and liberality Dr. Shattuck deserved well of the medical profession of his city and State and indeed of the whole country, and his memory is worthily perpetuated not only by the foundation of this lectureship but also by the endowment of the chair of pathological anatomy in the Harvard Medical School.

* Bulletin of The Johns Hopkins Hospital, 1899, X., p. 62.
† Published in Boston in 1808.

ARTICLE XIV.

# THE TREATMENT OF RETRO-DISPLACEMENTS OF THE UTERUS.

By CHARLES H. HARE, M.D.
OF BOSTON.

READ JUNE 12, 1900.

# THE TREATMENT OF RETRO-DISPLACEMENTS OF THE UTERUS.

THE subject assigned to me by your Committee to bring before you for discussion is an old one, but evidently most important judged by the time and skill daily utilized in trying to solve it practically. As yet no unanimity of opinion exists. One operator knows that tampons and pessaries never did any good; another knows that operations for simple uncomplicated backward displacements should not be done; another allows his favorite operation, but condemns all others. Alexander's operation, done hundreds of times by the same operator with success satisfactory to himself, has been said to be done but once a year by one of our greatest gynæcological teachers simply to show his pupils an operation which ought not to be done. The more operations there are for the same condition the more evident it is that no operation is satisfactory to all operators. The writer has found fifty-one operations and modifications of the original operation recorded for the treatment of backward displacements, but has no original one of his own to offer. The majority of these have been sifted out until only a few are advisable in the minds of those whose opinion has been obtained. In addition to retroversion and retroflexion this paper will consider briefly the retrodisplacement called retrocession or retroposition of the uterus which is usually if not always anteflexed with this malposition, and concerning which comparatively little has been written.

Concerning etiology there is much that we wish to know. Trouble follows certain well recognized causes, and yet the same conditions exist in other cases and give no trouble, and in still other cases no cause can be found for existing trouble. Certain it is that backward displacements are found accidentally from infancy to old age in the investigation of other conditions where pelvic symptoms did not exist. Soudrys in seventy-one autopsies on infant girls found nineteen backward displacements. Winckel found 10% of the uteri back in six hundred cadavers. In women complaining of pelvic symptoms Sänger found 15.43% due to retroversion and flexion; Winckel 19.10%; other investigators 17 to 18%. Adhesions follow labor, abortions, intra-uterine treatment, gonorrhœa, salpingitis, and unknown causes amongst which some writers class certain of the acute infectious diseases, while others observing the frequency of adhesions in strong and apparently healthy young women without other assignable cause, suggest the possibility of the pressure upon the rectum producing enough change in the nutrition of the pressed area to permit germs to pass through, cause inflammation and resultant adhesions. Schultze classifies causes as follows: (1) Relaxed ligaments from pregnancy, absorption after posterior parametritis, constipation and continued recumbent position; (2) Fixation of the cervix anteriorly by spontaneous and artificial scar tissue; (3) short anterior vaginal wall from undevelopment or senile atrophy; (4) habitual full bladder; (5) poor or torn perineal floor, permitting the anterior wall to press downward dragging the cervix forward and letting the fundus go back; (6) rarer causes: abnormally long cervix, tumors anterior to the fundus, adhesions of tubes and ovaries posteriorly. It is readily seen how easily improper dress, lifting and straining can help on when some of these causes start the trouble. Retrocession is claimed by some to be always due to a congenital shortening of the utero-sacral ligaments, while others

claim it is always due to an inflammatory shortening of these ligaments.

The question as to whether all backward displacements should be corrected is still disputed. Many cases undoubtedly exist without symptoms. Systematic examination of all cases one month after childbirth shows a considerable proportion of uteri back, and certainly some of these go on without discomfort, untreated, and also go through subsequent pregnancies without trouble, but many others are not so fortunate. Winter says 12% of primiparæ develop retroversion. Rörnheld, in 232 abortions, found 58% due to retrodisplacements. Many writers have emphatically expressed their belief that every retroversion and retroflexion, whether simple or complicated with some other trouble of the uterus or pelvic floor, sooner or later induces disease of the appendages. The majority of the best opinions state that we must expect congestion and changed pelvic circulation in all malpositions of long standing and that changes of nutrition will give changes of structure and some undesirable result, even though it may be delayed, until there follows a protracted climacteric with troublesome hemorrhages.

Attempts to correct retrodisplacements have been by tampons, pessaries, massage and operations. Massage has been most strongly advocated by Brandt and a few others, yet never commonly adopted. Rumpf, in an article in March, 1899, claims nearly all cases are symptomatically cured and that 50% require no other treatment. The bulk of the best opinions, however, are strongly against the utility and advisability of massage.

Pessaries have rightly in a great measure given place to operations. They have, like everything else, been misused and unjustly criticized. The writer believes they still have a proper place with some old women, with cases where some organic disease makes an operation inadvisable, and in such cases as refuse operation, or on fair presentation of

pessary and operation prefer the former with its objections. A cure cannot be expected from the pessary, but we can always try in cases of need. Davenport, in 1894, reported the ultimate results of the treatment of 584 cases of backward displacements with the pessary, and found fifty-two cures, or slightly less than 9%. Nine others had symptoms cured, but a return of the misplacement on removal of the pessary. One ultimate cure wore the pessary nine years with one failure during this time, on its removal too soon. In general his statistics showed an early cure, if cure was to follow the pessary, and that at least 75% must wear the pessary for life or have return of discomfort.

Prevention being the best cure, are we not paying entirely too little attention to these displacements in the case of puerpural women, where so many cases undoubtedly begin? According to Abels the critical period is at the beginning of the second week, when degeneration of muscular tissue has advanced but the connective tissue has not regained its elasticity and strength. Gottschalk advises examining and using the pessary if necessary on the seventh day after child-birth, keeping the woman in bed three to four weeks, lying nearly all the time upon the side, and urination every four hours. Possibly the interrupted electrical current might aid at the proper time. It was not long ago that the importance of the immediate repair of the perineum was recognized. Let us now try to correct some of our retroversions and flexions when they occur. The hard rubber Smith pessary or some modification of it is usually the best, as the vagina is normally narrower at the outlet than at the vault. The stem pessary is mentioned only to condemn it. No one would apply a truss to reduce an hernia. The same rule should follow the use of pessaries, which should not hold the fundus forward or up by pressure, but should hold up and back the posterior cul-de-sac, carrying with it the cervix, then intra-abdominal pressure keeps the fundus for-

ward. Dudley, in his Diseases of Women, says: "the pessary must be restricted to those cases in which the displaced organs are reducible, in which the pessary is capable of holding them in place, and can be worn without discomfort. Failure to recognize and appreciate the contraindications accounts not only for the failures and disappointments, but also for the many evil results which have followed the many indiscriminate attempts to treat all displacements by mechanical support. The exclusion of unsuitable cases, and the recognition of the necessity for accurate diagnosis are apparent. The pessary, according to the knowledge, judgment, and mechanical skill of the practitioner, will be useful, useless, or injurious." Within three months the writer has removed one Smith pessary which was bottom side up, another Smith pessary bottom side up, and also with the outer end at the vault; one Hodge pessary where there was an orange-size cyst crowding the uterus against the pubes, as proved by operation two days later. Comments are unnecessary.

To Alquié of France belongs the credit of first suggesting Alexander's operations, in a paper before the Academy of Medicine, in Paris, Nov. 17, 1840. The paper was referred to a commission which four years later reported its disbelief in the possibility of doing the operation and its doing good in case it could be done. The commission also commended Alquié and requested the Academy to thank him for not having performed the operation upon the living. Deneffe, in 1864, at Ghent, Belgium, was the first to try the Alexander operation upon a living woman and failed to find either ligament though opening both canals. Complaint was made and much trouble given him and his seniors for experimenting upon the living. Alexander was the first to successfully perform the operation which now bears his name, on Dec. 14, 1881, though Adams, not knowing this, did the same operation two months later.

Mundé, in 1884, first did the operation in America. Alexander's, like other operations, was not and is not perfection. Repeated modification of the method of finding and fixing the round ligaments have been adopted to overcome the faults of the operation. Edebohls, who opens the inguinal canal, then closes it, after the principle of the Bossini operation for hernia, was the first to pass the fingers through the internal ring to break up adhesions and do work upon the tubes and ovaries. Many have followed his lead with satisfactory results. Goldspohn, in May 1900, reports a most interesting series of ninety-five cases of adherent uteri and diseased appendages treated by inguinal cœliotomy. Smith, after pushing back the drawn-out peritoneum, fastened the cord with one silk-worm gut suture through the anterior wall of the canal and one half the ligament. Another suture includes Poupart's ligament, one half the round ligament and superior pillar of the ring. A third stitch, if needed, is used. Johnson, whom the writer is most apt to follow, pushes back the peritoneum, making a pad and completely closes the ring by a suture through the ligament and one side of the ring, another suture through ligament and other side of the ring and a third suture through ligament, bottom of canal and both sides of the ring. Kellogg, through a small incision, enters the roof of the canal at about its middle, hooks up and fastens the ligament. Goelet hooks the ligament by a small incision over the internal ring and secures by suture from the skin through edge of canal and by weaving the loop under the external oblique over the canal and folding across the bottom of the wound on the surface of the oblique. E. C. Dudley modified Kellogg's method by weaving the drawn out ligament into the tissues for an anchor. Cleveland pulled the end of the ligament under the fascia for an inch and a half in the pubes as an extra hold. Martin tied them under the pubic fat. There are still several other methods which we will not here mention.

Some of the best workers in gynæcology are opposed to Alexander's operation under any condition. Many, however, are not, and to this class the reader belongs, believing that in uncomplicated retroversion and flexion it is the best operation and that many of the objections raised have been excuses for a lack of necessary knowledge. Round ligaments have been reported as often absent, but anatomists tell us that the round ligaments are muscular tissue continuous with the muscular tissue of the uterus and are about as likely to be absent as the uterus itself, though exceedingly rarely they may go up and outwards from the internal ring. Johnson has operated and found the round ligaments, where a previous operator had tried the Alexander and reported the ligament absent. These ligaments have been found very small, but Newman has demonstrated upon the cadaver that any round ligament, however small, will bear more weight than is ever likely to be put upon it. The uterus is not suspended by the ligaments in proper cases, but is held forward. Penrose says: "No uterine ligament is inserted higher than its origin in the uterus, hence those ligaments do not suspend the uterus in normal position; the main factor is the retentive power of the abdomen or equality of pressure in all directions." The ligaments will sometimes be broken, but they can be picked up by opening the canal or, if necessary, the abdomen can be opened. Suppuration, with and without phlegmasia extending to the vessels of the thigh, has occurred, but with the asepis of to-day is rarely excusable. Pain, beyond some pulling for a time, does not occur, if the ilio-inguinal nerve is kept outside the fastening sutures. Many herniæ have resulted under the old methods of simply pulling out and fastening the cord. A very large per cent. of herniæ recurred after the old methods of operation for the radical cure of hernia, but such is not the case to-day. We shall not get herniæ after Alexander's, if we close the canal with

up-to-date methods. Alexander's has been called a difficult operation, but when one sees certain operators do it repeatedly in ten to twenty minutes, it can hardly be justly so classed, though the writer can, and no doubt the majority will always find a fundus through an abdominal opening more easily than a round ligament in the inguinal canal. Alexander's of to-day should have no mortality—though a few deaths have been reported in the past. The writer has found 2,526 recorded Alexander's by different operators, with sixty-two failures mentioned. The immense amount of work in following cases leaves the results with most operators unrecorded, and after spending much time in looking up and avoiding a second enumeration of the same case, the writer decided that these statistics are of little value in bulk. Of this number only 1,296 have pregnancy mentioned from the standpoint of age and marriage. Some accurately but many by guess; 135 pregnancies were reported. A few operators followed as many of their cases as possible most carefully, and their reports and statistics show no larger per cent. of abortions, discomfort during pregnancy, abnormal labors and retrodisplacements after labor than occur in unoperated women. In 1896, Johnson reported a most valuable analysis of 240 of his own cases.

In 1859, Sims suggested gastro-hysteropexy, without opening the abdomen, and prepared a canula and shaft with needle to pass a silver wire through the uterine canal and attach the fundus blindly to the abdominal wall. Ether was given the patient, but his courage failed and the operation was indefinitely postponed. Other men, however, did this later. The present idea of ventral fixation originated from the observation that extraperitoneal treatment of the pedicle of cystoma kept the uterus forward. Koeberlé, in March, 1869, was probably the first to do intentional fixation for a case giving symptoms of chronic intestinal obstruction. He removed the healthy ovary by

cœliotomy and sutured the pedicle in the lower end of the abdominal incision. Sims did the same operation in Feb. 1875, for a painful retroflexion with a small cyst in one ovary. Tait, in Feb. 1880, removed both ovaries, and in closing the abdomen passed one suture through the fundus of a retroflexed uterus for forward fixation. Hennig, in 1881, after removal of both ovaries, sutured round ligaments, broad ligaments and fundus to the abdominal wall for a retroflexion. Olshausen, in Sept. 1886, was the first really to describe by writing of ventro-fixation, as systematically done by him by attaching the round and broad ligaments near the cornu of the uterus to the abdominal wall with silkworm gut. In America, Kelly first read a paper upon hysterorrhaphy, in Nov. 1886, and described a case done in April, 1885, or nearly eighteen months before Olshausen's paper was published. Leopold later fixed the fundus to the abdominal wall — first scratching the fundus, causing no bleeding or as little as possible, then passing silk sutures through the entire abdominal wall and fundus, removing these sutures in twelve to fifteen days. From this time came multiple methods of placing sutures in one or another tissue and place, in the use of buried permanent and absorbable sutures, and in the development of other intra-abdominal methods of correction. In May, 1896, Noble, in a most valuable paper reported 808 cases of suspension by American operators in reference to pregnancy and labor, where at least one ovary had been left. He found fifty-six pregnancies; ten per cent. aborted; forty-three had been delivered, with one death. From French and German Journals, between 1891 and 1895, were collected 175 pregnancies, of which ten per cent. aborted; seven had premature labors, and eighteen had complicated labors. There were eight forceps deliveries, five versions, three Cæsarean sections and three deaths. The danger to pregnancy and labor was found to be an overdevelopment

and thinning of the posterior wall, while the anterior became imprisoned and hypertrophied. Noble then advised Kelly's operation, but with the attachment to the anterior of the fundus, which would be better obstetrically, though worse gynæcologically. Edebohls then said, split the Kelly and Noble methods and attach at the middle of the fundus. In 1898, Smith reported 2,500 ventro-suspensions with 148 pregnancies, thirty per cent. of which had abortions, painful pregnancies and operative labors. He found that suspension to the peritoneum only gave few relapses and no serious trouble during pregnancy and labor. For operation, then, he recommended scarifying both fundus and abdominal peritoneum and attachment of peritoneum to the anterior surface of the fundus with two silk sutures, and to prevent peeling passed one of the silkworm sutures closing the abdomen through the fundus which was to be removed in a month. Fowler has suggested dissecting out the urachus, cutting off one end and passing it through fundus like a ligature and then reattaching the cut end.

The round ligaments have also been utilized after opening the abdomen. In 1889 Polk reported catching the round ligaments three-fourths of an inch from the cornu and sewing them together in front of the uterus. In 1895, Mann, in case the abdomen needed to be opened for adhesions or other cause, advocated doubling the round ligaments twice upon themselves, making three layers of round ligament between cornu of uterus and pelvic wall, but did not recommend it where utero-sacral ligaments were very lax nor for prolapse. A. P. Dudley denuded the round ligaments, then sutured them together. Objections have been raised against these folding operations because of causing angles in the fallopian tubes. So far as known they have not been commonly adopted, though their originators reported excellent results. Bond, in 1898, stripped out at least three inches of the round ligaments after doing cœliotomy, pulling

these ends through part of the abdominal wall and tying them or suturing down the folds. Ferguson, in Nov. 1899, reported incising the abdominal wall to the linea alba, retracting the flaps, opening the sheath of the recti one inch from the median line, retracting the recti, then cutting through the transversalis fascia and peritoneum, seizing and cutting the round ligament a good inch from the cornu, pulling out this portion with some of the broad ligament suturing them to the transversalis fascia and peritoneum and carrying the end through or around the rectus suturing it to the rectus sheath. Gilliam, in April of this year, modifies Ferguson's method by going through the abdomen in the usual way, exposing the recti and pushing through them an inch from the median incision with some blunt carrier pulling back a loop of round and broad ligaments sufficient to flatten on top of the rectus and then fastening both in and on the top of the rectus. Morris, in April, 1900, reports opening the abdomen, seizing the round ligament, stripping back peritoneum from a median cut, suturing the loop, then recovering with the stripped back peritoneum. The same was done by Goelet by anterior colpotomy and reported in 1896. Kaan has just reported his method of puckering the round ligaments by a subperitoneal silk suture along the round ligaments, the ends of which pass through a little muscular tissue at the cornu and the ligament at the other end, then he puckers enough to bring the uterus into proper position and ties to hold permanently. Rather unique operations were those of Tormesco, who opened the abdomen and shortened the anterior uterine wall by removing a V-shaped piece, then sutured the gap to bring the fundus forward; and that of Spidler, who separated the bladder from the uterus, pulled the fundus towards cervix with a bowstring like suture, then fastening to the lower end of the abdominal incision.

The confusion in the use of the terms ventral fixation and

ventral suspension has made accurate separation of reported cases impossible, but classing them as best he could the writer has found 3,148 cases of ventral fixation reported. Of this number 2,971 are mentioned with reference to pregnancy, 355 cases being known which gave 70 abnormal labors. Of ventral suspension or attachment with sutures only to the peritoneum 624 cases were found without trouble reported during pregnancy and labor. In neither method did the per cent. of abortions reported seem above the percentage in unoperated women. Failures in anatomical result were more in suspension than in fixation.

Schücking, in 1888, described his method of vaginal fixation by blindly pushing a needle through the anterior wall to fix the fundus forward. Sänger, in the same year, mentioned the theory of anterior vaginal fixation with silver wire. Mackenrodt, in 1892, first reported his method of longitudinal incision of the anterior vaginal wall and curved incision in front of the cervix, separation of flaps and suturing the anterior uterine wall above the internal os to the vaginal flaps with two silk sutures. Dührssen reported a month later 114 cases and claimed priority of actual operation for his method of making a transverse incision in front of the cervix and attaching the fundus to the flap. In 1893 Mackenrodt and Winter reported failures and their modifications of the old operation by carrying the sutures as near the fundus as possible. In America, Vineberg, in January, 1894, first advocated vaginal fixation with slight modification of Mackenrodt's method, placing a silk suture one-half inch from edge of flap, three-fourths inch up from the urethra and through the anterior wall of the uterus near the fundus and a second suture an inch lower. Both Mackenrodt and Dührssen modified their operations later to vesicofixation or attachment to the peritoneum of the bladder. In May, 1897, Goffe reported his operation of shortening the round ligaments by folding each twice upon itself through

an anterior colpotomy, a transverse incision being made an inch below junction of vagina to cervix to permit the cervix to rotate back and a longitudinal incision from the centre of the transverse to the base of the urethra. At the same time Vineberg reported his method of seizing the round ligaments by anterior colpotomy three or four centimeters from the cornu and suturing this point to the lateral sulci, as near the pubic arch as possible. In 1898 Pryor reported his method of doing posterior colpotomy for adherent retroversion and flexion, then packing the cul-de-sac with gauze to create scar-tissue to hold the cervix back and up, thus keeping fundus forward, the claim being that adhesions from sterile gauze were not objectionable like those from inflammatory processes. Many subsequent changes of this gauze with strictest asepsis was needed, making it a very laborious method. Freund has corrected retroversion and flexion by sewing the utero-sacral ligaments to the posterior wall of Douglass pouch and obliterating the pouch with so-called aseptic inflammation by gauze packs. Mackenrodt, in 1898, reported his operation of vaginal fixation as good anatomically, but bad for labors, and advised as a substitute shortening of the broad ligaments by anterior colpotomy by approaching these ligaments in front with two or three sutures. Gottschalk last year reported some cases of intraperitoneal fixation through the vagina by fastening the anterior surface of the uterus to the utero-vesical fold of peritoneum. Dürhssen last year defended vaginal fixation as modified by him, reporting 352 cases with 2.3% recurrences and 23 pregnances with two forceps deliveries, once for long cervix and once for an old primipara. In May, 1899, Byford advocated Alexander's in nonadherent cases, but with adhesions advising anterior colpotomy for shortening the round ligaments and a suspension original with him by drawing down the fundus of the bladder, then suturing the fundus of the uterus to the post pubic peritoneum, which is

drawn down after the bladder, making it much like an abdominal suspension to the peritoneum and making an attachment of peritoneum to peritoneum instead of peritoneum to connective tissue as in the fixations of Mackenrodt, Dührssen and Gottschalk. The writer has found 826 cases of vaginal fixation recorded with 48 anatomical failures, 50 normal and 30 abnormal labors, but all abnormal labors were with the earlier methods of fixation. He has had no practical experience with this route and cannot justly criticize it, but theoretically prefers abdominal cœliotomy and ventral attachment where adhesions and appendages are to be treated.

Concerning retrocession little has been written. For the usual if not always associated condition of anteflexion all the operations devised for anteflexion have been done usually with some relief for a time, but with return of discomfort later. It seems yet to be decided whether the utero-sacral ligaments are congenitally short in retrocession or whether the trouble is due to a posterior parametritis which Schultze thinks is the cause of the majority of anteflexions. Papers by Burrage have most excellently presented retroposition with anteflexion. Massage is here allowable if ever, though the writer disbelieves in the forcible massage under ether as advised by Schultze. We can also try drugs, packing, douches, electricity, the lesser operations of dilating and curetting with or without drainage, posterior division of the cervix, Dudley's operation for straightening the canal, and at times an amputation of the cervix may be tried. These failing, as they are most apt to do, it is best to always combine a dilating and curetting, and if the case is extreme a Dudley operation or an amputation of the cervix with abdominal cœliotomy and cutting of the utero-sacral ligaments sufficiently to give the uterus free motion, then doing a ventral suspension preferably to the anterior of the fundus the better to aid intra-abdominal pressure to straighten the anteflexion.

The danger of opening the abdomen has yearly grown less, and the mortality of years ago is not honest argument against any operation to-day. It was the writer's misfortune last June to be chatting with a patient upon whom he had done ventral suspension eight days previously. Convalescence had been ideal, yet while telling me that she felt perfectly well she ceased to speak and died without the slightest warning. A careful autopsy by Dr. McGrath, showed a pulmonary embolus from a thrombus in the left internal iliac vein. Cultures from the peritoneum, embolus, thrombus, spleen, liver and kidneys, showed everything sterile. The writer cannot agree, therefore, with those who say the abdomen can be opened without any danger. An essential to all intraperitoneal operations for displacements, are adhesions or bands which are always pathological and repugnant to surgery, and only utilized because their use improves a much worse pathological condition. Herniæ after cœliotomy for displacements with present technique, should be exceedingly rare.

In conclusion the reader would express his belief that:

(1) Pessaries have a very limited but most useful field.

(2) Whatever the operation selected for the displacement, it is best to do all other necessary operations at the same time, such as curetting, plastic operations upon the cervix, vagina and perineum, and whatever is necessary to the tubes and ovaries.

(3) For retroversion and flexion without adhesions or trouble with the appendages, or a too lax condition of the utero-sacral ligaments, Alexander's operation with proper closing of the canal to prevent hernia, is the best operation, and is preferred by more operators than any one operation.

(4) In child-bearing women, where cœliotomy is necessary, suspension by Kelly's method is the best correction, but when past child-bearing period some form of fixation to the abdominal wall is surer:—closing the abdomen in

either case, where not contraindicated, by the layer method of continuous catgut in the peritoneum, interrupted catgut in the muscle, mattress suture in the fascia, preferably of silkworm gut or some other nonabsorbable suture, and any suture for the skin and fat.

(5) Where after opening the abdomen it is found necessary to remove both tubes and ovaries, remove the fundus uteri and the cervix also if indicated. If the woman is young save one ovary or portion of one, if possible, to prevent a stormy menopause, even though both the tubes need removing; in this case removing also the fundus and the cervix if indicated.

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ARTICLE XV.

# THE TREATMENT OF PROLAPSE OF THE UTERUS.

By WALTER L. BURRAGE, M.D.
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READ JUNE 12, 1900.

# THE TREATMENT OF PROLAPSE OF THE UTERUS.

PROLAPSE may be defined as a downward displacement of the uterus in which the cervix appears at the vulva. It is a species of hernia. The distinction between complete and incomplete prolapse is of little practical importance because the terms have a variety of meanings. For example, the uterus lies entirely outside the vulva, a condition which, strictly speaking, should be called complete prolapse, very exceptionally and in complete prolapse the vagina may or may not be completely inverted. Falling of the womb is a term popularly used to designate prolapse, and procidentia is usually applied to the more extreme forms. We must describe in each case the position of the uterus with reference to the surrounding structures; how much the os externum projects beyond the introitus vaginæ; the length of the cervix; the length of the body; the amount of eversion of the vagina; whether the bladder is dislocated and if so how much; the condition of the vaginal walls; whether the uterus is replaceable; the condition of the pelvic floor and so on.

The supravaginal portion of the cervix may be hypertrophied to such an extent that the cervix projects outside the vulva, while at the same time the position of the fundus of the uterus is little if any altered from normal. Such a conditon is clearly one of prolapse, although some authorities have maintained that it is not. On the other hand there is a certain amount of descent of the uterus in retroversion

and following laceration of the pelvic floor and weakening of the uterine ligaments from whatever cause. This condition is not commonly classed as prolapse.

The object of treatment in prolapse is to restore the uterus and vagina and bladder if dislocated to approximately normal positions, and to maintain them there by strengthening natural supports if possible, and, failing in this, to provide artificial supports.

At the outset it is important to have a clear conception of the anatomy of the structures and the nature of the forces concerned in this dislocation.

The position of the uterus in the pelvis under normal conditions is determined by its specific gravity, *i.e.* whether small and light or large and heavy; by the intra-abdominal pressure acting on it in all directions; and by its attachments to the surrounding pelvic organs and tissues.

Suppose a healthy nulliparous woman in the erect posture. The long axis of the uterus lies in the axis of the true pelvis with the fundus uteri touching a line drawn from the upper margin of the symphysis pubis to the promontory of the sacrum. The external os points towards the coccyx and is distant from it an inch, more or less. The long axis of the uterus is at right angles to the long axis of the vagina. When the bladder is full the uterus is retroverted and its axis approaches more nearly to the axis of the vagina. The uterus as it were floats in the peritoneal cavity. Although there is only a small amount of fluid in the peritoneum, under normal conditions the intestines with their fluid or semi-fluid contents filling the peritoneum, a closed sac, render the conditions about the same as if the peritoneum were filled with fluid. The position of the uterus is governed by the same rules of physics which determine the position of a body of given density floating in a closed vessel in a fluid of known density. Pressure is exerted on the body in all directions equally. If the body

is heavier it tends to maintain a lower level, and if lighter a higher level.

If the genital cleft, the pelvic floor and the vagina are in their normal state the bottom of the vessel is intact, so to speak, and the pressure on the different aspects of the uterus is the same; just as much up as down, just as much forward as back. If on the contrary the fat of the labia majora and thighs, which helps to close the vulva, is atrophied, if the pelvic floor has been torn and the vagina, instead of being shut is gaping, a column of air takes the place of the bottom of the vessel. Even then the abdominal contents and the uterus would not sink down if the abdominal walls and the diaphragm were rigid. You have all tried the experiment in hydrostatics of filling a tumbler with water level with the brim and then placing a piece of paper over it and inverting the tumbler. The water does not run out of the tumbler because it is held in by the pressure of the atmosphere, some fifteen pounds to the square inch, acting on the paper from below but not acting on the upper surface of the water because of the bottom of the tumbler.

The diaphragm and the abdominal walls represent the bottom and sides of the tumbler, but they are not rigid, and under certain conditions following pregnancy and in old age the abdominal walls lose their normal tonicity and become flabby, so that the retentive power of the abdomen is in a measure lost. Not only that, but the pressure from above may be increased by constant coughing, straining at stool, lifting heavy weights, by tight lacing, by enteroptosis or by ascites or abdominal tumors.

There are four sets of ligaments which steady the uterus in place much as the guys of a derrick prevent the derrick from falling. They are the vesico-uterine, the utero-sacral, and the round and broad ligaments, and to these must be added the attachments of the uterus to the vagina. Under normal conditions the ligaments act simply as guys and

not as supports. Under abnormal conditions they may be called upon to take a large portion of the strain upon themselves.

The uterus then may be said to be elastically suspended or floating in the pelvis, its mobility limited by the attachments of the broad ligaments along its sides, by the round ligaments and utero-vesical ligaments in front, and by the utero-sacral ligaments behind. The range of motion of the cervix is still further restricted by its attachments to the vagina. The uterus moves every time the patient breathes or takes a step and when she coughs and sneezes, and it is somewhat lower in the pelvis when the woman is standing than when she is lying down. It is not an uncommon experience to see a uterus which has formerly been prolapsed held in place by an attack of pelvic inflammation and the prolapse cured, only to return when the inflammatory exudate has gone.

Suppose the pelvic floor and perineum have been ruptured in childbearing, the vagina gapes and its walls are flabby, and its long axis has been carried backward several degrees by the lack of support from behind, there is a tear in the cervix and the uterus has not involuted as it should, and, being heavy, is low in the pelvis. The ligaments of the uterus have not regained their tone and the organ is retroverted, perhaps helped to this position by insufficient attention on the part of the patient to emptying the bladder. The uterine axis and that of the vagina coinciding, any extra pressure from above, and acting on the anterior or upper face of the fundus instead of upon the posterior face of the through fundus, gradually forces the cervix like a wedge down the vagina until a condition of prolapse is established.

It is important to note that a retroflexed uterus and an anteverted uterus cannot be prolapsed, lying as each does with its long axis at right angles to the long axis of the vagina, provided the pelvic floor is intact.

After the menopause, senile atrophy of the vagina, the vagina becoming shorter, tends to favor prolapse by the action of the vagina pulling downward on the cervix, while at the same time the disappearance of the fat in the labia majora and thighs opens the genital cleft, and the wasting of the abdominal muscles lessens the retentive power of the abdomen.

In certain forms of prolapse the cervix is enormously elongated with or without dislocation of the fundus. Such a condition is thought to result from prolonged traction by the vagina on a lacerated cervix associated with a certain amount of hypertrophy of the cervix due to chronic passive congestion. It is to be said, however, that this elongation of the cervix is found in the virgin and the nulliparous woman. The vagina is inverted to a greater or less degree in prolapse, generally carrying the bladder with it. There is some difference of opinion as to whether prolapse of the anterior wall of the vagina is the cause or the result of descent of the uterus, and it would seem that in some cases it is one and in others the other.

The rectum is seldom included in the prolapse anything more than a rectocele of moderate dimensions, but the peritoneum both between the bladder and the uterus, and between the rectum and the uterus generally follows the inversion of the vagina, and, in operating for prolapse we must remember that the peritoneal cavity as well as the bladder is often represented in the tumor. We should not forget that stagnation of urine and infection in a dislocated bladder sometimes leads not only to cystitis but to ascending infection of the ureters and kidneys.

In the consideration of the treatment of prolapse the most important portion is prophylaxis. In this connection a quotation from B. S. Schultze is not out of place. He says: "The puerperal state favors the origin of prolapse because in the first place it favors that of retroversion. A

very large number of retroversions may be traced by their history directly back to some puerperal condition premature or otherwise and retroversion recurring soon after confinement is far more often accompanied by descent than when it happens to a woman who has never been a mother, for all the attachments of the uterus have been relaxed by the tension, extension and change in the position incident to the gravid state. The sooner retroversion occurs after childbed, the more easily, on the average, is prolapsus uteri developed from it." ". . . . . in the puerperal state the prolapsed anterior vaginal wall may in some cases absolutely draw the uterus down out of its normal position. The wide, roomy and relaxed vagina is much more prone to prolapse soon after childbed, in any case where there has been a previous retroversion; the bladder, stretched out during pregnancy, is then relaxed also and, its contents being under the influence of intra-abdominal pressure, helps in the further depression of any uterus already lying in a position of retroversion with descent."

If we accept as facts these statements of a careful observer the conclusion is obvious, every woman should be subjected to a vaginal examination after the puerperium and a dislocated uterus supported by tampons or pessary, or the vagina, if relaxed, treated by astringent douches or suppositories and the amount of active and violent exercise limited until involution of the uterine organs has taken place.

Extensive injuries of the pelvic floor and cervix should be repaired with the object of assuring involution. It goes without saying that every effort should be made by careful management of labor to prevent such injuries. A physician's whole duty to his patient is not terminated when, following labor, his patient is up and about, she has no trouble with her breasts and the baby is well. He ought to know the exact condition of her uterine organs. It often takes five years or longer to develop a prolapse, which

causes annoying symptoms, from post puerperal injuries that might have been rectified in the months following labor.

In the treatment of an existing prolapse the first indication is the reposition of the organ in its normal position. In this way the passive congestion incident to the malposition is relieved, and ulcerated portions of the prolapse can best be treated.

The patient being in the dorsal position, the clothing thoroughly loosened, the bladder and rectum empty, the tumor should be carefully bathed with weak corrosive solution, anointed with vaseline and then grasped in the fingers of one hand. A little kneading and pushing will generally succeed in working it inside the vulva. In exceptional cases the knee-chest position is useful in making reposition. In the rare cases caused by the growth of abdominal tumors and by ascites and in those in which the uterus is limited in its mobility by pelvic inflammatory exudate, the prolapse is irreducible until the cause has been removed.

The uterus when first returned to the pelvis generally takes the position of retroflexion. By bimanual manipulation it should be placed in anteversion. Finally the vagina should be packed, preferably with dry cotton tampons dusted freely with boric acid powder. If ulcerations are present the packings should be renewed every second day. In other cases they may be left in place three or four days. Where the vulva and perineum are patulous a pad of cotton the size of a tennis ball should be placed on the vulva after the packing is in place, and held in position by a T bandage. If possible, the patient should maintain the recumbent posture and in any event every effort should be made to reduce the intra-abdominal pressure from above to a minimum, by treating a chronic cough or chronic diarrhœa and changing occupations which necessitate heavy lifting and straining. Tight lacing must be forbidden.

Various sorts of medicated applications may be made to ulcerated surfaces on the vagina by soaking the cotton of the packing with glycerine and ichthyol, glycerite of tannin, glycerine with iodoform, iodol or aristol, etc. When the ulcerations have healed and the vagina begins to lose the smooth, dry, integumentary appearance so characteristic of inverted vagina, we may begin to devote our attention to measures calculated to retain the uterus and vagina permanently in their normal situation.

Our task is made easier if the perineum still retains something of its former strength. In this event a well fitting Albert Smith or Thomas pessary will sometimes hold the uterus in place. More often it will not. Then we have recourse to the Gehrung pessary or the Meigs' elastic ring. Skene's cystocele pessary and the figure of eight pessary of Schultze have proved useful in my experience. Failing in the use of these we may try as a last resort a cup or ring pessary attached by a rubber tube to a belt around the waist.

Some patients, a decreasing number in these days, who are too old or too feeble to undergo a radical operation for the relief of prolapse, and many women in whom the prolapse has existed a comparatively short time, are made comfortable, and, in the latter class, permanently cured by the use of one of the forms of pessaries enumerated. Many of the intelligent patients may be taught to remove, cleanse and replace their pessaries themselves. It should be impressed upon the patient's mind by varied iterations that a pessary is a foreign body and must be frequently inspected and cleansed or much harm may result in the way of ulcerations from pressure. Some vaginæ are more tolerant to pessaries than others, but no pessary should be left in the vagina indefinitely.

In young women in whom prolapse has not existed too long, strong hope should be entertained of restoring the

natural supports of the uterus by palliative treatment, and we should not be in too great haste to advise operation as soon as we have made our diagnosis.

To summarize: The palliative treatment of prolapse consists in restoring the uterus to the pelvis and holding it as far as possible in its normal axis by cotton tampons until all ulcerations are healed and the vagina has regained a more healthy tone. At the same time efforts are made to reduce the intra-abdominal pressure from above by ordering the recumbent posture, removing tight clothing from around the waist and other measures, and finally, in attempting to support the uterus in its normal position by a suitable pessary, coming as a last resort to a pessary attached to a belt.

The radical or operative treatment consists of the following procedures:—

1. Repair of lacerated cervix.
2. Amputation of cervix.
3. Restoration of the pelvic floor and perineum.
4. Narrowing of the vagina.
5. Shortening of the round ligaments.
6. Attachment of the anterior face of the fundus uteri to the vagina or bladder.
7. Attachment of the posterior face of the fundus uteri to the abdominal wall.
8. Removal of the uterus.
9. Combinations of the above.

I. The first object in the operative treatment of prolapse is to get rid of the results of passive congestion and inflammation in the tissues of the uterus and thus insure involution and a diminution in the size and weight of the organ. To this end curetting and the repair of a torn cervix is indicated. Trachelorrhaphy should be preferred to amputation in young women and in cases where there is not excessive hypertrophy and induration of the lips of the torn cervix, because our object is always to restore to the normal

condition. The cervix has important functions in labor and a uterus without a cervix is more difficult to maintain in its proper axis. The usual Emmet operation fulfills every indication.

2. Amputation of the cervix is called for when there is present hypertrophic elongation of the cervix or when there is excessive enlargement or induration of the lips. The Sims operation has the best reputation but the Schroeder method of amputating is often useful. It is important that the uterine canal should be left large enough and should be covered with mucous membrane, both in amputating and in performing trachelorrhaphy, to prevent subsequent stenosis. It is my practice to do a curetting in every case of amputation and trachelorrhaphy, because of the endometritis which usually accompanies uterine misplacement of long standing.

3. The restoration of the pelvic floor and perineum accomplishes perhaps more than any other procedure towards the cure of prolapse. By it the normal axis of the vagina is restored and the patulous condition of the introitus vaginæ is done away with. The object sought is, as in the case of the cervix, to bring the structures to their original status except that in old women having atrophy of the tissues of the pelvic floor it may be allowable to unite tissues which were not intended to be united. That is to say, it may be advisable to unite the labia majora for the purpose of closing the genital cleft. Complete closure of the vagina for prolapse has been advocated and practised in the past by various operators and has been found unsuccessful, for subsequent to operation the tissues have stretched gradually and the prolapse has returned.

The operations on the posterior wall of the vagina and perineum which have given the best results in my experience are the Emmet and Hegar. In both most of the operation is in the vagina where the injury has taken place

rather than outside. If the lacerations in the pelvic floor have been in the sulci on either side of the vagina, the Emmet operation is preferable; if in the median line the Hegar, with a triangular denudation from just below the cervix to the fourchette, is to be chosen. The stitches should be passed deeply so that they catch up the sundered muscles and fascia. My preference is for interrupted sutures, part catgut and part silkwormgut; and the latter, the outside sutures, are left in position for from eight to ten days.

It is imperative after all plastic operations on the vagina for prolapse that the patient should maintain the recumbent posture for at least three weeks so that union in the tissues may be firm (experimental researches teach that it takes three weeks to obtain a solid uniting of apposed surfaces), and to avoid subjecting the newly joined structures to pressure from above. I am convinced that many failures are due to a neglect of this precaution and to the employment of operative procedures, such as the flap splitting operations, which draw the structures of the pelvic floor downward and outward instead of upward and inward where they belong.

4. Various measures have been adopted to narrow the vagina. Lefort's operation has been practised extensively abroad. It consists in uniting the anterior and posterior vaginal walls from the cervix to the ostium vaginæ by denuding a narrow strip on both walls in the median line and then bringing the two lines together by sutures. Thus the vagina is converted into two tubes lying side by side. The operation has manifest disadvantages as in no way approximating a normal condition. Lateral elytrorrhaphy, which has for its object the restoration of the upper exremity of the vagina and of the anterior vaginal wall to its normal location and direction has been advocated by certain gynecologists and has much to recommend it on theoretical grounds. In dislocation of the urethra downward, I have

found Skone's operation for stitching the tissues about the upper urethra to the subpubic ligament of value.

The essential point to be borne in mind in all operations on the anterior and lateral vaginal walls is that the object to be attained is the lengthening and stiffening of the anterior wall so that the cervix may be carried backward, the uterus anteverted, and the intra-abdominal pressure exerted on the posterior instead of on the anterior face of the fundus. The oval denudation on the anterior wall extending from the cervix to the lower portion of the urethra and closed by transverse interrupted sutures has about the best reputation. The Stoltz purse string operation is of doubtful value because it does not lengthen the distance from the cervix to the vulva.

5. Alexander's operation for shortening the round ligaments is not an especially valuable operation in prolapse for the reason that although it removes the slender portions of the round ligaments and raises the uterus as a whole in the pelvis it does not antevert the organ.

Shortening the round ligaments through the anterior vagina or shortening them through an abdominal incision has the disadvantage that the strong portions of the ligaments are shortened while the outer weaker ends have to bear the strain, and, as has just been said of the Alexander operation, shortening the round ligaments does not antevert the uterus. The results by these methods are said to be good in retroversion but they are of doubtful value in prolapse.

While we are considering the utilization of the natural supports and guys of the uterus for maintaining the organ in position and before taking up the more artificial measures, those that stitch the uterus to surrounding structures, it may be well to mention shortening the utero-sacral ligaments.

Stitching the cervix to the posterior cul-de-sac of the vagina has been resorted to as a means of holding the cer-

vix back, but as the vagina is far from being a fixed point in prolapse not much ought to be expected of this measure. The utero-sacral ligaments are much stronger than the round ligaments and have a more important office in anteverting the uterus. In Savage and Kiwisch's experiments to produce a prolapse of the uterus artificially the tension on the utero-sacral ligaments offered the greatest resistance, the prolapse occurring as soon as they were divided. Ought not we to make use of these ligaments if possible in the treatment of prolapse? In one case of prolapse I have shortened them through an incision in the posterior cul-de-sac of the vagina and the immediate and remote results were good. It is too early as yet to say much about the operation but it may be possible to shorten them successfully both through the vagina and through the abdominal incision. The subject is mentioned here rather as a suggestion than as one of the recognized methods of treatment.

6. The uterus has been held in anteversion in cases of prolapse and retroversion by stitching the anterior face of the fundus to the bladder or to the edges of the vagina after first performing anterior colpotomy. The vagina and bladder being as a rule dislocated and lax in cases of prolapse this method of operating would not commend itself, leaving out of account the fact that as done in the past the operation has been proved to be a cause of dystocia in subsequent pregnancy.

7. Ventral suspension or fixation as it was formerly called has a distinct value as an operative procedure in prolapse. It is not to be forgotten that this operation is not in any way to take the place of restoration of the natural supports of the uterus. It is a distinctly artificial measure. Nature never intended the fundus uteri to be attached to anything. It was to be free to enlarge with advancing pregnancy and involute after the expulsion of the child. Experience with the suspension operation has proved that the elastic liga-

ment which is formed between the parietal peritoneum and the posterior face of the fundus uteri, following properly executed operations of this sort, in no way interferes with subsequent pregnancy. The suspension operation is a failure in cases of prolapse when a heavy uterus is attached to the abdominal wall and no attempt is made to repair the pelvic floor and anterior and posterior vaginal walls and cervix. The chief value of the suspension operation is to direct the intra-abdominal pressure upon the back of the uterus. An anteverted uterus cannot be prolapsed.

In performing the operation I follow the technique of Howard Kelly except that I always use absorbable ligatures of chromicised catgut or kangaroo tendon and catch up enough of the transversalis fascia to prevent the parietal peritoneum from being stripped from the fascia subsequent to operation. If a suspensory ligament is sure to be formed, and we cannot, even if we wish, maintain the fundus permanently in close apposition with the parietal peritoneum, I fail to see the advantage of a non-absorbable ligature. The disadvantages of such a ligature are many.

8. Hysterectomy is called for in rare cases of irreducible prolapse where there is a suspicion of malignancy and in women who are near the menopause whose uteri are the seat of chronic metritis.

Hysterectomy alone without plastic operations on the vagina and pelvic floor fails to cure the prolapse. Some years ago, when the furor for abdominal operating came in, attempts were made to treat all pelvic diseases by this route, but from what has been said it is plain why prolapse cannot be cured by removal of the uterus. The uterus, to be sure, cannot prolapse, but the vagina, bladder and abdominal contents can. Some success has been attained by attaching the vagina to the stumps of the broad ligaments after hysterectomy. Hysterectomy by itelf cannot be considered a leading operation in the treatment of prolapse,

because prolapse generally includes dislocation of the vagina and bladder.

9. It is plain that there is no one operation which is a specific in prolapse. There are many obstinate cases of long standing prolapse in old women with lax abdominal walls, atrophy of the vulva, perineum and vagina, in which permanent cure is a difficult matter:—whence the diversity and multiplicity of operations invented for the relief of this disease. In cases of recent origin I have obtained good permanent results from trachelorrhaphy and anterior and posterior colporrhaphy, the patient perhaps wearing a pessary for several months subsequent to operation to antevert the uterus. In cases of long standing I have added a ventral suspension to the operations just enumerated with good late results. All the operations are usually done at one sitting. To summarize: Treat prolapse by palliative methods if there is a reasonable prospect of ultimate success. If not, aim to restore the uterus to its normal size and shape, and by repairing the injuries of the pelvic floor and perineum by painstaking plastic operations endeavor to reestablish the proper relations of the uterus and vagina, adding abdominal operations and the more artificial measures only in the extreme cases of long standing.

ARTICLE XVI.

# THE OPERATIVE TREATMENT OF UTERINE DISPLACEMENTS BY VAGINAL FIXATION.

## (DÜHRSSEN'S METHOD.)

By WILLIAM M. SWIFT, M.D.,
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READ JUNE 12, 1900.

# THE OPERATIVE TREATMENT OF UTERINE DISPLACEMENTS BY VAGINAL FIXATION.

I HAVE ten cases to report. The operation done is practically the one described and practised by Prof. Dührssen of Berlin. The parts are shaved, washed with green soap, scrubbed and douched with a lysol solution, one drachm to the quart. In my last operations I have worn rubber gloves and found they did not interfere with the necessary manipulations.

The cervix is dilated and the uterus curetted with a sharp curette; afterwards with a douche-curette, and washed out with sterilized water. The bladder is emptied with a glass catheter. The anterior lip of the cervix is then seized with Orthmann's fixature; the uterus is brought down as far as possible and held forward. An incision about half an inch long is made through the vaginal mucous membrane at the upper border of the anterior lip of the cervix. This incision is prolonged with scissors about half an inch on each side. A double tenaculum is put on the vaginal mucous membrane at the centre of the incision, and traction made upwards. The bladder is then separated from the uterus by the finger, aided at times by the closed points of the scissors. It is important to carry the dissection well out on each side so as to have the uterus as well as the bladder out of the way. By keeping close to the uterus there is no danger of wounding the bladder. This dissection is carried as far as the internal os. It is a mistake to strip off the peritoneum from

the uterus farther up than this point. The fold in the peritoneum may be felt by the finger against the anterior surface of the uterus. It should be seized with two pairs of forceps, and opened with scissors between them. The bladder is then dissected away from the peritoneum still farther; the peritoneum slit up, and a suture put in each side for a guide. A pair of double hooks are put on the vaginal wall about an inch below the urethra. The middle point of the vaginal wall in the transverse incision is seized with forceps. Traction is made with both instruments, and the ridge made is slit up with scissors. The triangular flaps of vaginal wall formed by the transverse and longitudinal incisions are then dissected away from the bladder. They usually peel off easily with the shut points of the scissors. This incision gives a wound extending from the cervix nearly to the urethra, and laterally about an inch on each side of the cervix.

The examination of the uterus and adnexa is next made. The uterus is then seized as high up as possible with stout, double hooks. The fixature is removed and the cervix pushed back with the speculum. The uterus is then delivered through the wound, any adhesions being separated at the same time with the finger. When the uterine tissue is soft the hooks may tear out several times before the uterus is finally delivered. The tears should be at once sewed up with a fine needle and cat gut. No ill effects come from them so far as I have seen. This gives ready access to the uterus, tubes and ovaries either for their removal or the excision of diseased portions.

After whatever is indicated has been done, the fixation sutures are passed through the uterus, one opposite the insertions of the round ligaments, and another about half an inch below. The sutures are then passed through the peritoneum and vaginal wall on each side. In four cases I have passed the fixation sutures around the base of the round

ligaments and have got good results. I think it safer, however, to pass them through the uterus itself. For the fixation sutures I use cat gut and find it satisfactory. In my first two cases I used silk worm gut, but it cut the tissues and was difficult to remove. The uterus is then replaced. The wound in the peritoneum is sewed up carefully with a continuous cat gut suture. If this be well done, no raw surface is left and the adhesions holding the uterus forward will only be between the two layers of peritoneum. The wound in the vagina is then sewed up with a continuous cat gut suture. Any operation on the cervix, vagina or perineum may now be done.

In my ten cases two had complete prolapsus, eight retroflexion. In only one case were there no adhesions. In two, both tubes and one ovary were removed. In one, both tubes and ovaries. In two, one ovary and tube.

In two, cysts of both ovaries were punctured and curetted. In three, amputation of cervix. In two, anterior vaginal wall narrowed.

In three, perineum repaired. In four cases, fixation sutures were passed around the bases of the round ligaments. In six cases, fixation sutures were passed through uterus near the fundus.

In two cases an abscess formed and discharged spontaneously through vaginal wound. This was before rubber gloves were used.

In all cases, except one, the subjective results were good. In the exception, both tubes and the right ovary were removed, the left ovary was allowed to remain. The patient was much relieved for a time; afterwards complained of pain over left ovary when menstruating. Hysterotomy was done on this account by another surgeon. Ovary said to be cystic. In no case was there any bladder disturbance. Six cases were examined three years after operation. Two, one year after operation. Two, two months after operation.

In all cases the uterus was found to have retained the forward position. In eight cases an enlarged uterus had become normal in size. In all the cases the uterus was freely moveable. In no case was the vaginal scar tender.

Where vaginal section can be easily done, there can be no question that it is much to be preferred to the abdominal route, so far as the immediate effects of the operation are concerned. A small vagina makes the operation difficult even with lateral incisions. The uterus may be so large that it fills the whole vaginal wound when it is delivered. This makes it impossible to get at the adnexa without removing the uterus. A fixed uterus, that cannot be drawn down at all, makes this operation impossible. Such cases as I have reported are not suitable for shortening the round ligaments outside, and the question presents itself, which is better, vaginal or abdominal section? The chief advantages of the vaginal method are:

1. That it is a less serious operation than abdominal section.

2. There is no abdominal scar and risk of hernia.

3. There is hardly any handling of intestines and less consequent danger of intestinal adhesions.

4. The patient has much less discomfort immediately after the operation. She can turn at once and is usually up in two weeks.

5. The ease with which any operation can be done on the tubes and ovaries, after they are delivered.

6. Any operation that is indicated on the cervix, vagina or perineum may be done without changing the patient's position.

7. In case there is infection the pus finds a ready outlet through the vaginal scar.

Dührssen reports 349 cases with no mortality where the operation was uncomplicated. In this number there were eight relapses; forty-four patients became pregnant; seven

abortions, four of which were brought on artificially. There were three cases of premature birth, one at eight months and one at six months. Four cases of forceps, three said to have been easy. Eighteen normal cases at full term. Dührssen states that he has had twenty cases of normal pregnancy and labor where the peritoneum was carefully sewed up. Schauta reports seventeen cases of vaginal fixation, Dührssen's method. Eleven cases report relief of symptoms; one had symptoms recur; one had severe hemorrhage. Two showed no improvement. In ten cases an examination was made, in all except one case one year after operation. In one case six months after the operation. In eight cases the result was perfect. In one case the uterus was in the middle position, and in one case there was recurrence.

He reports pregnancy in eight women. One woman aborted, time unknown. Another aborted, first pregnancy, went however to term later and had normal labor. One woman was thrice pregnant and had normal labors and living children. One woman at the time of examination was four months pregnant. The remaining four women required to be delivered. One case of forceps, one placenta prævia. In two women there were complications due to previous operation. Martin and Gottshalk of Berlin, Vineberg of N. Y. and Kober of Breslau have reported favorable results.

Vaginal section and fixation has not become popular in this country. One objection made is, that it is unsurgical, as the surgeon does not see what he is about. This is not true if the uterus be properly delivered. Bladder disturbances and tenderness of the vaginal scar are also spoken of. I have not seen these symptoms. The chief objection to the method is, the possible interference with subsequent pregnancies and complications with labor. This may be still a mooted question, but the evidence that Dührssen brings

forward is strong. It certainly must make a very great difference in the character of the adhesions whether we sew up the peritoneal wound carefully or let the uterus become adherent to a broad, raw surface. The argument that the operation is difficult ought not to influence us. In properly selected cases the operation gives perfectly satisfactory results. It is always well to have the patient prepared for abdominal section. In a number of cases where I have expected to operate through the vagina, I have decided to do laparotomy after putting the patient under ether. Emergencies may arise at any time during the operation that require abdominal section.

Dührssen: Die Einschränkung des Bauchschnitts duch die Vaginale Laparotomie.
Schauta: Monatsschnift fur Geburtsshulfe und Gynækólogie, Jan., 1900.
Martin: British Medical Journal, Jan. 4th, 1896.
Wilson: British Medical Journal, Feb. 26th, 1898.
Vinberg: New York Medical Journal, Oct. 27th, 1894. American Medico-surgical Bulletin, March 7th, 1896. American Journal of Obstetrics, July, 1897.
Webb: Lancet, August 23d, 1897.
Gottschalk: Centralblat, fur Gynækologie, No. 4, 1899.
Kober: Centralblat, fur Gynækologie, August 20th, 1898.

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## DISCUSSION.

Dr. R. H. Seelye, of Springfield.—May I suggest one point that has not been brought out sufficiently in the papers or discussion? I have been disappointed that it has not been brought out in this meeting, and have been disappointed in my results, and that is, in regard to procidentia in fat and aged women. It has been my misfortune to come in contact with several patients who have been fat and old and who have had procidentia. I have performed, I think, all of the operations and made use of all the methods that have been suggested, except the vaginal fixation, with absolutely no good results whatever. I have done ventro-fixation and hysterectomy with no good result. The hysterectomy has been followed by prolapse of the vaginal walls.

| History. | Examination. | Operation. | Immediate Result. | Ultimate Result. |
|---|---|---|---|---|
| Mrs. S. B., 27 years. Two children, youngest one year. Menstruation irregular and painful. Pain constant in left side. | Patient anemic. Large retroflexed uterus very tender. Mass in region of left ovary. | Aug. 22d, 1897. Vaginal section, uterus retroflexed and bound down by adhesions easily broken up. Left ovary and tube prolapsed and adherent. Right ovary cystic. Left ovary and tube freed from adhesions. Right tube and ovary removed. Uterus fixed by ligatures passed on right side through stump of tube on the left around the round ligament near its insertion into the uterus. Peritoneum sewed up with cat-gut. | Patient did perfectly well. Passed urine on second day and walked out in two weeks. | Seen May, 1900. General condition good, able to do work. Menstruation not regular without pain. Uterus normal in size and position. Micturition normal. Vaginal scar not tender. |
| Mrs. L. R., 21 years. Probably gonorrhea two years before. Since that time pelvic pain and marked pain at menstrual period. Since marriage pain worse. Flowing most of time but not profuse. Coitus so painful impossible. | Uterus much enlarged, retroflexed, very sensitive. Enlarged tubes on both sides. | Operation Aug. 1897. Uterus large, retroflexed. Extensive adhesions over fundus of uterus and tubes. The left tube and ovary firmly adherent. The tube disorganized and occluded. It was removed. Left ovary normal. Right tube and ovary much diseased, both removed. Uterus fixed by three silkworm gut sutures through uterus itself. Peritoneum sewed up. | Three years after operation. Uterus perfect position. Small. Some tenderness over left ovary. Relief from pain except at menstruation, then pain over left ovary. Micturition normal. | Last winter vaginal hysterectomy done, by another surgeon. Uterus found in normal position. Left ovary cystic. Indication for operation, pain at time of menstruation. |
| Miss E. B., 36 years. Much pain in lower part of abdomen. Pressure in pelvis. | Uterus large, retroverted. Somewhat fixed, tender. | Aug. 1897. Uterus large, retroverted. Slight adhesions. Tubes and ovaries normal. Fixation sutures passed through uterus, cat-gut. | Patient passed urine morning of second day. Did perfectly well and was up in two weeks. | One year after operation. Patient's condition good. Micturition normal. Pain relieved. Uterus much smaller. Normal position. Vaginal scar not tender. |
| Mrs. A. S., 48 years. Prolapse for 25 years. Since birth of first child. Worse for two years. Suffered great deal. Considerable hemorrhage and not been able to work. | Complete rupture of perineum and tear into rectum for about two inches. Complete prolapse of enlarged uterus. Cervix enormously hypertrophied and ulcerated. | Sept. 1897. Vaginal section. Tubes normal. Both ovaries contained small cysts, punctured and curetted. Fixation sutures passed around the basis of round ligaments. High amputation of cervix. Large flap of vaginal mucous membrane cut away on each side to narrow the vagina in front and the perineum repaired. | Patient did well in every respect. Kept in bed longer than usual on account of prolapse. | Seen May, 1900. Relief of symptoms. Does all her work. Control of spincter. Vagina small. Uterus high up and small in normal position. Vaginal scar not tender. Micturition normal. |
| Mrs. M. N., 37 years. Six years before at birth of child had perineum lacerated to sphincter. Since two years complete prolapse. Not able to work. | Cervix much hypertrophied. Complete prolapse. Perineum ruptured to spincter. | Sept. 1897. Vaginal section. Fixation sutures passed round the bases of round ligaments. High amputation of cervix. Vagina narrowed in front and perineum repaired. | Patient did perfectly well. Kept in bed on account of prolapse. | May, 1900. Result perfect. Patient does a great deal of work. Vagina small and uterus high up in normal position. Vaginal scar not tender. Micturition normal. |
| Mrs. L. M., 26 years. Abortion at two months. | Uterus very large, flabby. Curetted. Flowing stopped. Uterus very tender and retroflexed. | Sept. 1897. Two weeks after curetting. Vaginal section. Retroflexion. Uterus delivered with some difficulty owing to its size. Small cysts in both ovaries, punctured and curetted. Tubes normal. Fixation sutures passed at basis of round ligaments. High amputation of cervex. | Patient did perfectly well. Passed urine on second day. Up in two weeks. | One year after operation patient aborted at 4 mo., was curetted at that time. Patient has been in much better general condition since operation. Micturition normal. At present patient 4 mo. pregnant. Uterus in normal position. Vaginal scar not tender. |
| Mrs. M. A. L., 28 years. For several years much pelvic pain. Most in left side. For one year pain very severe and constant. Much pain with menstruation. Much leucorrhœa. | Uterus large, retroflexed, tender and somewhat fixed. Mass on left side tender. | May 24th, 1899. Uterus large, retroflexed, quite firm adhesions. Cyst of left ovary size of small orange. Right ovary cystic. Both tubes and ovaries removed. Vaginal fixation through uterus. | A week after operation patient had a rise of temperature and chill. In a few days pus was discharged through the vaginal wound. Afterwards patient did well. | May, 1900. General condition good. Never felt so well in her life as at present time. Menstruation regular, but rather slight. Very little pain on left side. Micturition normal. Uterus small, normal in size and position. Vaginal scar not tender. |
| Mrs. A. F. Ill for two years. Last four months much pain in iliac region and back. Menstruation painful, flows two days, stops, flows again. | Uterus retroflexed, movable. Left ovary enlarged. | May 19th, 1899. Uterus normal in size, retroflexed. Left ovary cystic. Removed. Vaginal fixation through uterns. | Patient had pus discharge through vaginal wound. Afterwards did well. | May 8d, 1900. Anemic, complains of some general pain, and feeling tired. Menstruation rather irregular. Marked gain in weight. Micturition normal. Uterus movable, normal in size and position. Vaginal scar not tender. |
| Miss C. D. Since last July much pain in left side, also pain in vagina. Pain constant, not much more when menstruating. | Uterus retroflexed, adherent, much tenderness. | Feb. 5, 1900. Uterus retroflexed, adherent. Both tubes much diseased. Removed. Left ovary cystic, removed. Right ovary normal. Vaginal fixation through uterus. | Patient did well. Passed urine on second day. Up in two weeks. Micturition normal. | May 25th, 1900. Much better. has very little if any pain, except when menstruating. Menstruation painful and not much flow. Bowels constipated. Uterus normal in size and position. Scar not painful. |
| Mrs. S. S. For several years pain in back. Last six months much worse so that patient could not work. | Uterus large, retroverted and fixed. Lacerated perineum. Lacerated cervix. | Mch. 29th, 1900. Uterus large, somewhat adherent. Retroflexed. Vaginal fixation through uterus. Amputation of cervix. Perineum repaired. Hemorrhoids removed. | Patient did perfectly well, but was kept in bed on account of perineum. | Examination made May 15th. Much less pain. General condition improved. Micturition normal. Uterus normal in size and position. Vaginal scar not tender. |

Ventro-fixation and suspension have, by virtue I think of the pendulous abdomens, been torn away in all cases. I should very much like to know the result of the experience of some of the gentlemen present. What can be done in such cases?

Dr. W. L. Burrage, of Boston.—It has not been my experience to have such cases as Dr. Seeley speaks of. I have seen old and fat women with prolapse. These are the difficult cases of course, on account of the amount of fat everywhere, and, the tissues of the pelvic floor having atrophied, there is less chance of restoring them to their normal condition. I think these cases are cured if the plastic operations are properly done and the other operations added to them; that is, the suspension operation or the fixation that Dr. Elliot speaks of. A more solid attachment of the uterus to the abdominal wall should be made in women who have passed the menopause. Dr. T. A. Emmet claims that he gets perfect results without any abdominal operation. The essential point is the manner in which the plastic and not the abdominal operations are done.

Dr. J. W. Elliot, of Boston.—I do not know that I can do better, in discussing this subject, than to give you my methods of treating these cases. Although I have rather gone into general surgery and gone out of gynecology, still I find that I have a number of cases of retro-version which are still treated by pessaries. There are a certain number of young, athletic women who bear pessary treatment very well, and who recover permanently after not a very long time. One point in this pessary treatment that I have discovered is that it is essential to begin with the very smallest pessary possible. If you do not, you stretch the ligaments, you stretch the vagina, unnecessarily. I consider that I have not begun a case properly if my first pessary is not too small. I want to know, in the first place, how small a pessary will hold the uterus, and you can never know that until you put in one so small that it will not hold the uterus. If the first pessary is too small you can easily increase the size. It is not very long, even with the small pessary, before I try to diminish it. I do not go many months without trying a smaller one, and in that way I have

had a surprising number of cures without any operation. I can remember many patients, healthy young women, who have been cured by pessaries.

I find, among other exercises, that horseback riding is very valuable for retro-version. It requires a smaller pessary if a patient is riding than if she is simply sitting about in a saggy chair, which bends her spine.

Of course when a patient is neurasthenic, so that the pessary treatment taxes her nervous strength, or if there are evidences of beginning neurasthenia, I do not attempt pessary treatment. If such a patient comes to me I always advise an operation at once. I do not put her through a long treatment of pessaries. Also, if there is a tender ovary, or if the pessary is not well borne, patients are not treated by pessaries but by operation.

The Alexander operation I have practically given up. In the first place, because it takes longer to do it. Then the bruised fat about the rings is liable to suppurate. In spite of the best care almost every one gets sepsis occasionally. I am sure that in certain fat and flabby individuals if a little piece of fat is included in a ligature, simply the germs that come from the air are enough to cause suppuration. For this reason, and for the reason that when it is done the uterus is not in its normal position, I have practically abandoned the Alexander operation.

I have, in a number of cases, shortened the round ligaments from the inside. It is an easy operation and, I think, a safe one. It doubles the weak parts of the ligaments. You are able to see the ovaries and tubes, and the damage done is very slight.

The operation that I do most for retro-version is Kelly's operation for ventro-suspension. I always do ventro-suspension in young patients and in women who expect to bear children. Where there is no expectation of bearing children I do what might be called ventro-fixation, which consists, as Dr. Burrage said, in fastening the posterior surface of the uterus to the abdominal wall, making the intra-abdominal pressure come on the back of the uterus, so that it cannot retrovert.

In prolapse, after doing the vaginal operation, I sew the back of the fundus of the uterus to the abdominal wall. I sometimes shorten the round ligaments at the same time

merely to have one more thing to hold. I do not believe in hysterectomy in prolapse. I consider the uterus useful in holding up the other parts. If the uterus is taken out when the fascia is torn away there is nothing to hold the bladder up. In spite of sewing up the vagina, the bladder will roll down. I once did a hysterectomy for a fibroid, which was prolapsed, and the prolapse continued after the operation and was even worse. The cervix I left on account of the nature of the case. The cervix pushed down and became œdematous just as if the uterus had been there. I believe keeping the uterus in as long as possible is of advantage in these cases. In regard to vaginal fixation I have had no experience, but I do not favor it.

In the first place, we have to consider, in recommending an operation to ourselves and the profession in general, what the chances are of the technique succeeding. Now in laparotomy, the technique with the Trendelenburg position is absolutely perfect. There is no danger of any contamination which is not provided for, well understood and foreseen. The light is perfect, the opening is very small; you are ready for every emergency. By the vaginal route you have to contend with the excreta from the bladder and the rectum. The folds of the vagina must be kept clean, which is a very difficult thing to do. Beside that the parts are sensitive and the patient is not always placid about being scrubbed and taken care of in the most thorough manner, and I always feel that there is danger over which one has no control. I always feel so when I have even a drainage tube in the vagina.

On the other hand I see no objection to a small opening in the abdomen in any of these cases. There is no danger of hernia in these operations where the opening is small. I do not think I ever had any hernia after ventro-fixation.

As I have never seen the operation of vaginal fixation, I do not know in exactly what position the uterus is left, so I have no right to compare the physical success of the operation. From my own inclination, my own experience, the other way is safer, clearer and more exact.

Dr. E. L. Twombly, of Boston.—I would like to ask Dr. Elliot one question. He spoke of the value of horseback riding in retro-displacements. I would like to ask what he thinks about bicycle riding in those cases.

DR. ELLIOT.—I will say in a general way that bicycling and golf have been pressing questions for the last few years. It it a difficult thing to answer in an individual case. I can simply say that, on general principles, I believe in all of the new out of door exercises, especially for women with retro-version. I believe in the new woman and girl as regards physical condition. I believe in their doing everything out of doors that they can possibly do. I advise it unless I can see some definite harm. I do not know just what bicycling does to a retroverted uterus. As far as riding a horse goes, I never suggest riding to any one who has any inflammation of any sort either in the tubes or ovaries.

DR. SWIFT.—I think what Dr. Elliot has said is true. It is impossible to make the vagina and surrounding parts as aseptic as in abdominal section. I remember hearing Landau say one could do anything through the vagina without trouble. I think that even in a very short abdominal incision there is danger of trouble from the inside. I have hardly ever seen the inside of an abdominal wound where there were not adhesions to the intestines and omentum. I think these adhesions are important.

In treating backward displacements of the uterus I have found the knee-chest position of a great deal of benefit in women who had a heavy uterus and often the result is quite surprising. I advise taking this position for five minutes at a time twice a day.

ARTICLE XVII.

# CARBOLIC GANGRENE.

## GANGRENE PRODUCED BY THE APPLICATION OF DILUTE SOLUTIONS OF CARBOLIC ACID.

By FRANCIS B. HARRINGTON, M.D.,
OF BOSTON.

READ JUNE 12, 1900.

# CARBOLIC GANGRENE.

## GANGRENE FROM THE USE OF DILUTE SOLUTIONS OF CARBOLIC ACID.

THAT dilute solutions of carbolic acid applied to the extremities for a number of hours may produce gangrene and total destruction of the part is a fact which the public at large and even many physicians are ignorant of. It is an unfortunate result of the popularization of the antiseptic treatment of wounds. Carbolic acid, once the favorite antiseptic among surgeons and now discarded, has become a general household remedy for the treatment of slight wounds and bruises. During the last five years, at the Massachusetts General Hospital, I have discovered myself, or have seen through the courtesy of the out-patient surgeons, eighteen cases of gangrene from this cause. In a large proportion of these, amputation has been necessary. These, together with cases which I can find in the medical literature of various countries, make a total of one hundred and thirty-two cases of gangrene from the application of dilute solutions of carbolic acid. It would be safe to assume that many hundreds of fingers have been destroyed from this cause. Doubtless many cases occur and are not recognized as due to carbolic acid; and these, with many others, are never reported. Usually the patient has come to the Hospital with the story of having treated a cut, a bruise or a felon with a solution of carbolic acid, which has been purchased from an apothecary for the purpose. Occasionally the treatment has been suggested by a physician.

The facts are these: — An aqueous solution of carbolic acid (1 to 5%) if applied to an extremity, as the fingers or toes, for a number of hours in the form of a moist dressing or poultice, may produce gangrene and total destruction of the part. This result is not from tight compression but simply from the action of the carbolic acid.

The following history of a case illustrates the occurrence. The patient came to the Out-Patient Department of the Massachusetts General Hospital. A delicate girl, age twenty-six, cut the tip of her right index finger with a piece of tin. Her brother, a strong, healthy man, had treated a cut finger two weeks before with a solution of carbolic acid which he had bought from an apothecary. His cut healed quickly and without trouble of any sort. She adopted the same treatment, washed the finger with the same solution and put on a bandage which she saturated with the carbolic solution. This was 6 P.M. On going to bed she moistened the dressing again with the solution. There was some pain in the finger during the night. In the morning, on removing the dressing, the skin of the finger was gray in color and the finger felt "lifeless and heavy." It was somewhat swollen throughout, and especially at the base. The color changed in a few hours to a dark brown. The discoloration was general and extended nearly to the base of the finger; this being the limit of the bandage.

A poultice of flax-seed meal was applied. The numbness remained. When the finger was allowed to become dry it became quite black. Various dressings were applied of a soothing nature.

At the end of four weeks, when I first saw the finger, it appeared to be in a condition of advanced gangrene. (See Plate I.). It was clay-colored in places, dark brown in some, and black in others. The nail appeared bluish black. A well-marked "line of demarcation" had formed near the

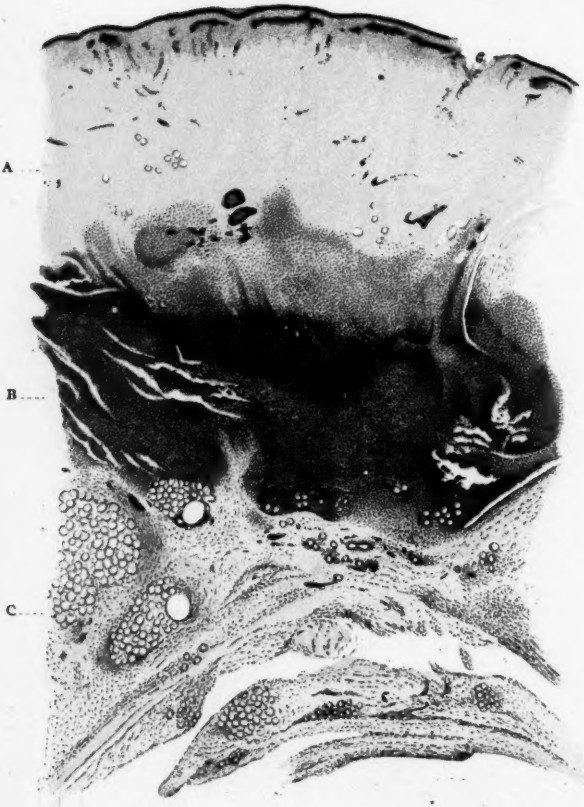
PLATE I.

CARBOLIC GANGRENE.

Appearance of a finger four weeks after the application for twenty-four hours of a dilute solution of carbolic acid. The finger was wrapped in cloths which were saturated with the carbolic solution not stronger than five per cent. Amputation necessary. Inflammatory process at the base of the finger shown by the reddened tissues.

Microscopic appearance shown in Plate II.

PLATE II.




CARBOLIC-ACID NECROSIS. Low power.

A. Area of total necrosis (diffuse red staining) with numerous colonies of micrococci (dark blue).

B. Area of purulent infiltration, diffuse finely granular blue staining from the large number of cells.

C. Area of round-cell infiltration. Fat and fibrous tissue with abundant cells and thrombosed vessels in outer zone.

See Plate I.

end of the first phalanx. The superficial tissues of the remainder of the phalanx were reddened and swollen. The finger was amputated at the middle of the first phalanx. Dr. William F. Whitney made the following report upon the microscopic appearance of this amputated finger:

"A transverse section was made completely through the finger after removal of the bone. This was mounted on celloidin and stained with polychrome methylene blue and eosine. The skin and subcutaneous tissue stained diffusely, with the exception of numerous large colonies of micrococci, which were found in the breaks on the surface and could be followed into the deeper layers. The vessels in this area were thrombosed and colonies of bacteria were present in the blood clot. Separating this from the deeper layer was a wide zone of round cell infiltration, among which polynuclear leucocytes were present in large numbers. In places the tissue was liquefied and there was a distinct formation of pus. From this to the periosteum there was a diffuse infiltration of round cells, and many of the small vessels were thrombosed with areas of hemorrhage among the tissues."

"The process may be regarded as a total superficial necrosis, with deeper purulent inflammation and hemorrhage." (See Plate II.)

This record is so like the history of other cases observed in the Out-Patient Department of the Massachusetts General Hospital that a report of them would be a needless repetition.

The history of the reported cases varies but little. The patient suffers from a wound or pain in the extremity, and by advice, sometimes unhappily of a physician, sometimes of a friend, and often because of his own idea of the proper treatment of such conditions, he purchases from a pharmacist a solution of carbolic acid. The member is wrapped with a dressing saturated with the solution. If the solution is strong enough and the length of time of its application is long enough, the result will be a loss of the part. It is sometimes impossible to determine the exact strength of the solution which has been used. It is safe to assume that it



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is not stronger than a five per cent. solution, since water, which is the common solvent, will take up only about 5% of the acid. Numerous cases (see Lévai (33) and others) have been reported, in which 3% and 2% solutions have caused gangrene which has resulted in amputation.

Peraire (41) reports a case in a child of ten years, who lost the second and third joint of the finger after twenty-four hours of exposure to a compress saturated with a one per cent. solution of carbolic acid. It is, therefore, plain that any solution of carbolic acid between one and five per cent. is dangerous. It is needless to add that gangrene does not always follow the use of carbolic solutions in this manner. In the report of the case occurring at the Massachusetts General Hospital, which I have given in detail, I mentioned the fact that the brother of the girl, who lost her finger by carbolic gangrene, had previously used the same solution in the same manner with no bad effect. The result depends on the strength of the carbolic solution, the manner of application, the length of time of application and the power of resistance of the individual.

The method of application commonly employed is to wrap the finger or the toe in cloth and to saturate this with the solution. Usually, in the cases reported, these wrappings have been kept saturated for from twelve to twenty-four hours. It is probable that the strength of the solution has less to do with the unfortunate result than the length of the application and the thickness of the individual's epidermis. Women made up four-fifths of the cases appearing at the Massachusetts General Hospital.

The destructive effect of pure carbolic acid is generally recognized, and sufferers from the cause are now rarely seen except as the result of accident. Moreover, the injury from pure carbolic acid is usually less serious than from weak solutions. The action of weak solutions is insidious. The injury is done without causing suffering. Strong car-

bolic acid, as Lévai (33) has pointed out, forms a scab which resists penetration of the carbolic into the deeper tissues, so that complete gangrene and destruction of an extremity is less likely to follow from the use of liquefied carbolic acid than from weak solutions.

The following experiment was made with the consent of the patient, an elderly woman, whose arm was to be amputated for sarcoma of the elbow.

Aqueous solutions of carbolic acid from one to five per cent. were applied to the fingers which had been covered with gauze. The gauze was saturated, each finger with a different strength. Unfortunately for the result of the experiment, the gauze was not kept saturated through the night. When the compresses were removed they were found to be perfectly dry and the skin beneath them entirely unchanged. There was not the least maceration of the skin nor any apparent difference in the appearance of either finger. Had the compresses been kept moist throughout the night it is probable that a certain amount of gangrene would have resulted. The maceration of the skin, which results from the moist application, is a very important element in allowing the penetration of carbolic acid. It might be expected that a thick epidermis would withstand the action of carbolic solutions longer than a delicate one.

In 1871 Tillaux (55) reported three cases of carbolic gangrene. In 1877 J. D. Kellock (25) reported two cases in the Canada *Lancet*. Since that time medical writers in various countries have reported cases and called attention to the danger of the use of carbolic solutions. In 1888 M. Kortum (27) thought that the gangrene was produced by the action of the carbolic acid upon the trophic and vascular nerves of the part. In 1890, Frankenburger (13), by experiments upon animals, proved that dilute solutions of carbolic acid would produce complete destruction of the parts to which they were applied.

As a result of microscopic study of the tissues he concluded that the carbolic acid acted directly upon the red and white corpuscles of the blood, producing stasis and thrombosis, thereby preventing all nutrition, and he believed that this condition produced death of the parts. He thought that the effect upon the nerves was of minor importance.

In 1896, Joseph Lévai (33) reported twelve cases of carbolic gangrene, the result of dilute solutions, observed by himself, and mentions fourteen other cases which he had seen resulting from strong carbolic acid. He collected reports of forty-two cases of carbolic gangrene. His observations led him to doubt the theories of Kortum (27) and of Frankenburgen (13), that the gangrene was produced by the peculiar action of carbolic acid upon the nerves or upon the blood vessels. By a series of careful experiments he shows that the death of the part is due to a direct chemical action on all parts of the tissues. He shows that carbolic acid has no specific quality for the production of gangrene, but that other diluted chemicals might produce the same effect. Five per cent. solutions of muriatic acid, nitric acid, sulphuric acid, acetic acid, and of caustic potash produced gangrene as well as carbolic acid, when applied to an extremity, by a moistened compress for twenty to twenty-four hours.

"The histological examination shows that in the beginning each of the diluted chemicals applied in the form of a moist dressing produces the same effect; the epithelial layer becomes œdematous and loosened." "As soon as a way has been made to the deeper layers each agent produces in a different way the same result—namely, necrosis of the living tissues." The death of the part takes place in layers downwards, and affects not only the nerves and vessels, but all the tissues at the same time. Maceration of the skin having taken place, as a result of the prolonged action of

# PLATE III.

FIG. 1. Thumb after the use of a dilute solution of carbolic acid. Amputation necessary. Thumb was scored in the attempt to save it. Case of Dr. C. L. Scudder.

FIG. 2. Appearance of finger of a young woman twenty-five days after the use of a dilute solution of carbolic acid.

FIG. 3. Appearance of a finger twenty-seven days after treatment with a dilute solution of carbolic acid for a crush of the last phalanx. The solution had been purchased from an apothecary who said it was five per cent. Amputation at the end of the first phalanx. Case of Dr. C. A. Porter.

FIG. 4. Result of the use of a weak carbolic-acid solution for a felon. After a number of weeks the last phalanx dropped off, leaving the above appearance. Case of Dr. Scudder.

FIG. 5. Finger after the use of dilute solution of carbolic acid for slight injury. Amputation necessary at line of demarcation.

FIG. 6. Same as Fig. 2 after amputation.

FIG. 7. Appearance of finger three weeks after the use of a weak solution of carbolic acid for paronychia. Amputation necessary.

the watery solution, the penetration of the chemical becomes easy and rapid.

Experiments and observations show that the gangrene does not result primarily from tight bandages, although tight bandaging undoubtedly increases the tendency to this process. The gangrene is limited to the parts enveloped by the moist compress, for in some cases, as mentioned by Lévai (33), the last phalanx of the finger, when not covered by the dressing, has escaped, and may continue to have its natural appearance and to bleed when pricked for some time, when the finger nearer the trunk may be quite black and without sensation. The gangrenous process may sometimes be so slight that only the skin is destroyed. Dr. C. A. Porter was able in such a case to save in part the usefulness of a finger by skin grafting before cicatrization took place. The treatment of this condition must vary according to severity of the process. In many cases it soon becomes evident that amputation will be the only helpful treatment. If the process seems superficial and the case is seen soon after the removal of the carbolic dressing, it might be beneficial to apply a dressing saturated with a bland alkaline solution, such as lime water.

It has been asked why gangrene does not occur upon the trunk. Superficial gangrene does occur if the application is sufficiently prolonged and the saturation of dressings is great enough. The result is not so disastrous because of the greater thickness of tissues and because the blood supply cannot be shut off as it is in an extremity. It is the enveloping of the entire part, as a finger or toe, with the dressing which causes the complete destruction of the part, since the gangrene only affects those parts which are thus covered.

The public must be taught to use some safer treatment. Moist dressings are often very soothing and helpful in slight injuries. A large part of the benefit to be derived

from any form of moist dressing can be obtained by using boiled water on clean compresses. Safe household remedies for this purpose are tincture of hamamelis or solutions of borax or of boracic acid.

It is evident that carbolic acid solutions in any strength applied as a moist dressing is dangerous and ought never to be used. The fact that it is often used without bad results renders it the more dangerous. It is the duty of the medical profession to see that this needless destruction of fingers is stopped. Carbolic acid of any strength should be included in the list of those drugs which can only be procured by a physician's prescription. Whatever the strength, it should always be labeled as dangerous.

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ARTICLE XVIII.

# SUPPURATIVE PERICARDITIS AND ITS SURGICAL TREATMENT.

By CHARLES B. PORTER, M.D.
OF BOSTON.

READ JUNE 12, 1900.

# SUPPURATIVE PERICARDITIS AND ITS SURGICAL TREATMENT,

## WITH AN ANALYSIS OF FIFTY-ONE CASES REPORTED IN LITERATURE.

Your Committee invited me to present the subject of suppurative pericarditis because they knew that in 1895 I had had a successful case of pericardotomy in a patient of Dr. F. C. Shattuck, who asked my surgical aid in its treatment. The case in full was reported to the American Surgical Association in 1897, together with a study of the literature of the subject up to 1895 and 1896. As all here may not be familiar with that report, I should like to read two short extracts from it, and then present the literature of the subsequent years with additional cases and some new ideas.

In my previous study of the subject I collected twenty-four published cases, beginning with the first case of recovery, the case of Rosenstein, operated upon in 1881. Of this number eight recovered and sixteen died, and in one the result was unknown.

In one case pericarditis developed one month after a wound. Many of the cases were complicated with left empyema, and twice the pericarditis was discovered at operation while draining the empyema. Of the fatal cases, two died at operation; one lived two days; one (Delorme's case) lived eight weeks after the operation.

The organisms found in the exudate were the staphylococcus aureus and streptococcus pyogenes, pneumococcus, and colon bacillus (in case of stab wound).

The amount of pus evacuated varied from ten ounces to two quarts. In one (Dickinson's case), it was brownish and thin with arterial blood; in two (Korte's and Eiselberg's cases) foul and thick; in two (Newman's and Delorme's) thin. In the majority it was thick, creamy, without odor. In a number of cases large fibrinous masses were found, the pericardium being covered with thick layers of lymph. At autopsy in one fatal case (Pepper's), the whole sac was full of a thick mass of fibrin as large as the fist; the drainage had been good, and the heart probably

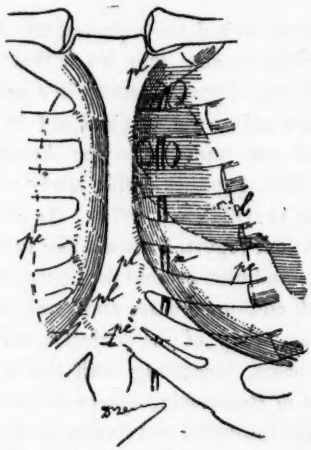

FIG. I.—Normal contour of Pleura and Pericardium (Delorme and Mignon). pl, pleura; pe. pericardium; l, border of lung.

failed from pressure of these fibrinous clots. The shortest case of healing of the sinus after drainage was nineteen days, the longest two months.

Preliminary aspiration was done in all these cases before opening the pericardium. In a number of cases paracentesis was done more than once, with and without injection of carbolic acid; and incision was finally resorted to owing to the rapidity of the reaccumulation. The immediate relief to pulse and respiration was almost always striking.

## THE SURGICAL ANATOMY OF THE PERICARDIUM.

All authorities agree as to the great variations in the line of reflection of the pleura and pericardium.

Sick found that in the adult, out of twenty-three cases the pleural reflection at the level of the fifth rib cartilage lay either at or within the left border of the sternum in seventeen; at the level of the sixth cartilage the pleural border had not gone beyond the sternal border in ten; at the level of the sternal articulation of the seventh cartilage it was in nine cases at the sternal border, or this cartilage was below its lower border. Twice it was less than one centimetre from the sternal border.

In twelve children Sick found at the level of the fifth rib cartilage the pleura was either within or just at the sternal border in eleven. At the level of the sixth cartilage the pleura had not left the sternal border eight times.

It will thus be seen that, accordng to Sick's careful observations, even at the fifth space the reflection of the pleura will often be behind the sternal border. Brooks (quoted by Quain) in four of seven quite healthy cases, found the left pleural reflection entirely behind the sternum, and in one at the sternal border.

According to Luschka (quoted by Quain) the pleura normally diverges from the median line at the upper border of the fifth costal cartilage, so that at the level of the fifth cartilage it is one-fifth centimetre, at the sixth two centimetres, and at the seventh 3.5 centimetres, external to the left border of tbe sternum.

Delorme and Mignon found in thirty-two adults that in the fourth interspace the left pleural border was within the border of the sternum in seventeen. At the level of the fifth cartilage it lay fifteen times internal to the sternal border, and seventeen times outside. In twelve cases at the fifth interspace the pleural border was at, or inside, the sternal; and in the sixth space the pleural border was out-

side the sternum in twenty-six cases, and at or within it in eight.

Dr. Thomas Dwight, Professor of Anatomy at Harvard University, agrees in the main with Sick's observations, but states that there are many variations, and that frequently it is possible to reach the pericardium through the fifth intercostal space and frequently not. Owing to the fact that the sixth intercostal space is small and narrow, and that even here the pleura often reaches the sternal border, he concurs

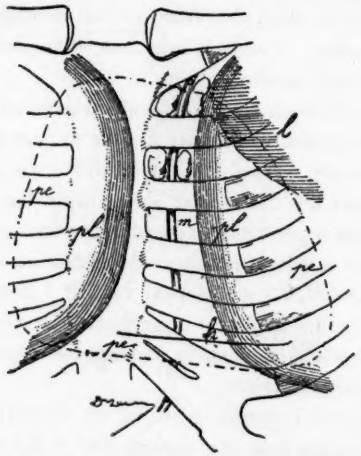


FIG. II.—Line of Pleura, abnormally to left of sternum (Delorme and Mignon). pl, pleura; pe, pericardium; l, border of lung; h, base of heart.

with the writer in advising resection of the fifth costal cartilage, and if necessary the excision of a piece of the sternum opposite this cartilage.

The internal mammary artery, according to Quain, runs parallel to the sternum at a distance from it of one centimetre. Delorme and Mignon, in thirty cases, found it a distance from the sternum of from one-half to two centimetres, the distance averaging about the same in the first six interspaces.

In looking over the arrangement of the left pleura by Dwight, Delorme, Sick, Quain and Testut, all agree that there is a varied arrangement. Below the fourth intercostal space in the majority there is a slight interspace close to the border of the sternum which is free from pleura. In twenty-two operations on the cadaver by myself it was found that the removal of the fifth intercostal cartilage, and the removal of half an inch of the sternum opposite the sterno-costal joint, gave free access to the normal pericardium near its lowest level.

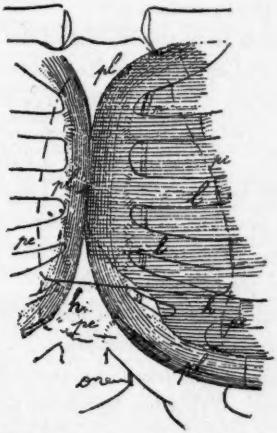


FIG. III.—Left Pleura, behind Sternum, reaching beyond middle line (Delorme and Mignon). pl, pleura; pe, pericardium; l, border of lung; h, base of heart.

Three of the methods of operation which have been proposed and practised, namely, trephining the sternum (Riolan), approach through an intercostal space, and epigastric incision (Larrey), should all be discarded, as it will be evident from the above account of the anatomy that in all of them there is danger of wounding the pleura or diaphragm.

In this earlier work I decided that operation was indicated in all cases of purulent pericarditis and perhaps in serous pericarditis in cases where aspiration once or twice repeated

is followed by reaccumulation of the fluid; and the following "ideal operation" was planned after careful consideration and experiment on the cadaver in order so far as possible to meet the following indications:

(1) To avoid opening the pleural cavity. This may be made more easy by adhesions as a result of tapping or inflammation.

(2) To open the pericardium opposite the point where drainage will remain good after the sac has contracted.

(3) To secure permanent and free drainage.

The steps of the operation are:

(1) An incision from the middle of the sternum outward over the fifth costal cartilage to its junction with the rib.

The soft parts are cleaned from the cartilage with periosteum elevator, care being taken not to wound the pleura on the under surface. The cartilage is divided with bone forceps from the rib and the sternum. The internal mammary artery and vein are thus exposed, ligated in two places, and divided between. The triangularis sterni is separated from the sternum and pushed to the left.

A little careful dissection with the director in case fat is encountered, exposes the pericardium, which is normally much thicker than the pleura. An aspirating needle should now be introduced, if this has not been previously done, in order to corroborate the diagnosis. If confirmed, the knife should follow the needle. The incision in the pericardium is best made obliquely downward and outward, beginning close to the excised border of the sternum. The edges of the pericardium should be stitched to the soft parts.

Irrigation should always be employed, with the object of removing any masses of fibrin which may lie at the bottom of the cavity; and if there are many such masses, it should be continued until the fluid returns clear.[1] The fluid may be weak sublimate or carbolic solution, or salt solution, according to the preference of the operator. The fluid must

be warm, and must have free exit.[2] With this exception no harm has resulted from irrigation, which has been practised in more than half the cases.

Drainage is best provided by two rubber tubes, one long and reaching to the bottom of the sac for the inflow, and a short tube just entering the sac for the outflow. As the discharge diminishes one tube may be removed, and finally gauze drainage inserted. Gauze drainage has proved adequate from the first, but where the fluid is thick or flocculent, tubes give the only adequate facilities for the subsequent daily irrigation.

The after-treatment must, of course, be directed to two ends: first, systematic treatment, consisting of forced feeding and free stimulation, and second, the care of the wound and the maintenance of drainage. The wound should be irrigated daily and the patient, if his strength is sufficient to allow it, turned on his stomach to facilitate drainage.

Roberts of Philadelphia, also, before the meeting of the American Surgical Association in 1897, presented an elaborate and exhaustive review of this subject. In this report he advocates the method of turning up a flap consisting of portions of the fourth and fifth left costal cartilages with the attached soft parts, thus exposing the field of operation. This method he had never followed on the living subject. Roberts collected thirty-five cases from all literature up to June, 1897, including my own case.

The previous work of the writer and the careful paper by Roberts brought the collected knowledge of this subject up to the summer of 1897, and leaves us nothing further to do than search the work of the last three years, and make our final conclusions and analyses.

Voinitch-Sianojensky of St. Petersburg, in 1897, a Russian writer in the *Annales de Chirurg. Russe*, 1897, xiii, ii. f. 3; also *Revue de Chirurgie*, 1898, vol. 18, p. 993, contributed a long study on the anatomical questions

in pericardotomy, but added nothing new to the conclusions of Delorme and Mignon, or to the anatomical studies of the writer given above. He refers to a case of incision in the second intercostal space for suppuration in the pericardium done by Bexman of Russia in 1891, with recovery. This case is not mentioned, so far as known, elsewhere. He refers to a number of different methods of incision, and the advocates of each as known to him.

(Through the third intercostal space—Sievens.

Through the cartilage of the fourth rib—Eiselberg.

Through the fourth space—Rosenstein, Orlow, Minine, Bronner, Parker, Klefberg.

Through fifth rib cartilage—Ollier, Gussenbauer and Körte.

Through fifth space—West and Davidson.

Desault and Robinson, as well as himself, advise operation through the sixth rib and sixth space.

Del Vecchio resects fourth and fifth ribs and cartilage, also Roberts.

Delorme and Mignon the fifth and sixth; Riolan, Læennec, Skielderup and P. Malle propose to trephine the sternum in the median line.

Velpeau, Pirogoff, Baizeau, Dolorme and Mignon think it unnecessary to keep to left side of the sternum, while Desault, Romero, Karanæff, Trousseau and Tillaux advise it. Rotch, Wilson and Dickinson chose fifth space on right side for tapping.)

He claims to have had the opportunity of studying post-mortem twenty cases of pericardial effusion, and to have done one hundred pericardial operations on the dead, and on this experience he declares that incision through the sixth costal cartilage will *always* avoid the pleura (which is not an accurate statement), and his proposed method of operation is to go through the sixth cartilage, and, if necessary, cut away some of the seventh cartilage also.

He states that open incision in all cases of pericardial effusion (not only in purulent cases) should be much preferred to puncture, because of the danger of wounding the heart and pleura by any method of using a needle.

Eichel, in 1899 (Die Schussverletzungen des Herzbeutels, Archiv. f. Klin. Chir., Band 59, Heft 1, 1899), (also Annals Surgery, vol. 30, p. 658, 1899), gives a fairly complete resumé of the anatomy of the pericardium and pleuræ with several diagrams. This article is chiefly valuable as showing that the most modern anatomical work does not change the accepted views of 1897 as given by me. In his paper he makes reference to all the latest foreign anatomics. (Lehrbuch der Topographische Chirurg. Anatomie von Joessel und Waldeyer, Bonn, 1899; also Terrier et Raymond, Surgery of Heart and Pericardium, Paris, 1898.)

He recommends pericardotomy for all effusions as well as for all wounds. He gives several interesting cases of operation for wounds and resulting hemorrhage into the pericardium.

The use of tubes for drainage after hemo- or pyo-pericardium operations Eichel opposes, stating that he had a case in which he could not keep the ends of the tubes from impinging on the heart wall and causing stormy and irregular action. He says that Riedel had a case that taught him the same thing (ref. to Riedel–Verhand der Deutsche Gesellschaft für Chir. 26th Cong., 1897, S. 72), also (Centralblat f. Chir., 1897, p. 56).

A point which is of great importance, and which Brentano and Schaposchnikoff lay great stress upon—viz., the anterior situation of the heart in all effusions into the pericardium, is denied by Eichel. In his case, he says, there was plenty of fluid between the heart wall and the anterior wall of the pericardium.

Brentano (Deutsch Med. Woch, 1898, 32, p. 506) reports five cases of operation for pericarditis, and discusses

this question as to the situation of the heart when the pericardium is more or less full of fluid, whether it is nearer to the posterior than the anterior wall. From his clinical experience Brentano decides that in all pericardial effusions the heart is situated down and forward, and most often lies right against the anterior wall of the sac even when no adhesions exist, and that in many cases there will be adhesions between the pericardium and the anterior wall of the heart. In all cases most of the pericardial fluid is behind. If this anterior situation of the heart is a fact, the danger of wounding the heart wall in all cases of paracentesis is great.

Brentano advises open incision in any effusion, with resection of the fifth costal cartilage. In fact, this seems to be the growing thought in the last few years, that incision through resection of the fifth left costal cartilage is a far safer operation than paracentesis.

Schaposchnikoff, in 1898 (Russisch. Archiv. f. Pathologie Klin. Med. und Bacteriologie von Padw. July, 1896; also Deut. Med. Woch, 1898, No. 38, p. 611), agrees entirely with Brentano on this question. He states that he has studied the point for twenty-seven years clinically, post mortem and experimentally, and has proved that the heart is in all cases forward against the sac wall.

Brentano recommends almost the identical procedures of my own technique, viz., stitching of the sac to the skin, and irrigation; for drainage, however, he always used iodoform gauze, not tubes.

He decides that opening the pericardium through a resection of fifth rib cartilage is very simple, and advises against puncture in any case. Local anæsthesia with cocain is all that is needed in most cases. In regard to this question Körte—speaking at the Freie Vereinigung des Chirurg. at Berlin, Dec. 13, 1897 (Deut. Med. Woch. 1898, p. 170)—favored the use of cocain in many cases, but stated that in certain cases of thick chest walls, ether

or chloroform must be used. Brentano mentions as one of his chief arguments against puncture in cases of serous effusion that it is impossible to remove all of the fluid by aspiration alone. Of 80 cases of paracentesis tabulated by West, in 23 the puncture had to be repeated.

Brentano reports five cases which are as follows: All five were done by resecting the fifth rib cartilage. Two were for purulent pericarditis, both of these cases following acute osteo-myelitis in children seven years old. Operation was followed by temporary and marked improvement, but death occurred in twelve days from pyemia, as shown by autopsy.

In both cases there were found multiple abscesses in the heart muscle itself, and in one the direct cause of death was from rupture of one of these abscesses into the left ventricle.

Two cases were for sub-acute sero-fibrinous pericarditis after rheumatic attacks and rheumatic endocarditis. Both died in three days.

The fifth case was a brilliant case of open incision for simple serous pericarditis, without preliminary puncture.

These cases are given in full in the list of cases appended to this paper.

Delorme, in 1897 (Gaz. des. Hopitaux, 1898, p. 1150), the same surgeon who collaborated with Mignon in 1895 in an article on the technique of pericardotomy (Revue de Chir. September and October 1895), recommends resection of the fifth rib cartilage to relieve mediastinal and pericardial adhesions whether or not due to tuberculosis, and also as the route to the pericardium in all cases.

H. Allingham (Lancet, Mar. 10, 1900) brings forward an entirely new method of operation for draining the pericardium in suppurative cases, and also, with Ogle of London, reports a case of purulent pericarditis operated on, though not by his suggested method. The case was one of purulent pericarditis originating from a chronic abscess in the pleural cavity near the pericardium. Incision after

resecting the fifth cartilage was followed by death in fourteen hours.

(This case is given in my list of cases.)

Allingham's suggested method which he has tried on twelve cadavers, but never on the living, is as follows:—It is designed to give best access to the sac and best dependent drainage by opening the sac from below through the diaphragm.

1. An incision about three inches long with its upper end at the costo-xiphoid angle is made along the lower edge of the seventh costal cartilage; the latter is then exposed by separating the abdominal muscles from it, the cartilage can then be pulled up and back, exposing the fibres of the diaphragm together with the cellular interval between its attachments to the cartilage and to the xiphoid appendix.

2. This cellular space is enlarged by cutting or tearing through the muscle of the diaphragm as far as necessary, when a mass of fat is usually seen just above the diaphragm in the space between the diaphragm below, the sternum in front, the pericardium above and behind. This fat, together with the diaphragm, is then pulled down when the pericardium presents, and can be opened up at its lowest point.

During the operation the peritoneum may be exposed to a slight extent as it sweeps downward from the under surface of the diaphragm. It is, of course, not injured, being pushed away as in a supra-pubic cystotomy.

The advantages claimed for this method are as follows:

1. The pleural cavity cannot be injured, as it is far away in the normal arrangement of the organs and further off in pericardial distention. It is claimed that this is the only sure method of avoiding the pleura.

2. Drainage is through the most dependent part of the sac, through a large opening not limited by bone or cartilage.

3. Great ease of exploration and cleaning of the sac is afforded.

In certain fat subjects it may be necessary to cut away some of the seventh cartilage to get room in the xiphoid space. (It is to be noted that Roberts has recommendod this area as the point of election for puncture, though not for incision).

Ljunggren (Nord. Med. Ark. New Series, 1899, vol. ix. No. 28) extracted briefly in Annals Surgery, 1890, vol. 30, p. 659) reports a case of suppurative pericarditis successfully treated by incision and drainage. (The full details of the case will be given in the list of cases.)

He objects to puncture as a dangerous procedure in any case, recommending incision and drainage as the rational thing in all cases. He added 6 cases to Roberts' 35, making 41 in all, but 4 cases are inaccessible to us and doubtful. Of these, 16 recovered and 25 died. Of the 16 recoveries, 8 were complicated by other diseases; in 7 of the fatal cases death was due either to faulty methods or slowness in operating.

His eight rules for the technique of the operation do not differ from my published method. He uses rubber drainage tubes, double.

The pleura should be bluntly loosened and sutured laterally to prevent infection of the pleural cavity. He mentions the occasional necessity of cutting away some of the sixth cartilage after resecting the fifth, in order to get more room, but does not mention removing any of sternum. General anæsthesia is not necessary and in weak cases is contraindicated.

Lilienthal of N. Y., in 1889 (Journal American Med. Assoc., 1899, vol. 33, p. 1422, also Med. News, Nov. 25, 1899) reports a case of recovery from operation in a boy convalescing from lobar pneumonia; operation under eucain, local anæsthesia, forty oz. pus evacuated, giving cul-

tures of pneumococci; this organism and streptococci were also in the sputum. (Details of this case are given in the list of cases.)

C. Mansell-Moulin, in 1897 (Trans. Clin. Soc., 1897, vol. 30, p. 217) reports an operation for "hæmopericardium" as he calls it. The case was successfully operated on about one month after a blow on the chest at foot ball, and six pints of thin dark bloody fluid removed, from which no cultures were made. It seems reasonable to class this with purulent cases like certain other cases of suppurative effusion after injury (note the case of Riedel, also case of West). The case is not very dissimilar from Eiselberg's at the first aspiration.

Sevestre in 1898 (Lancet, 1898, April 23) reports a case of purulent pericarditis in the course of acute pneumonia. Cultures, pure pneumococcus. Operated on under cocain anæsthesia. Double empyema afterward with resection of ribs on each side. Death. (See list of cases.)

In Sevestre's case, two aspirations were attempted before the incision was finally resorted to. At the second aspiration only bright blood was obtained (the query arises as to whether the heart itself was wounded); the operation through the fourth space seems to have been careless, and the empyema following may have been caused by the pericardial operation.

H. Bethan Robinson, who reported his first case in Trans. Clin. Soc., vol. xxx., has reported a second case, in 1898. (British Med. Journal, 1898, Nov. 26, vol. 2, p. 1605.) A case following broncho-pneumonia in a child four years old. Death in three days. (See list of cases.)

Several recent elaborate papers given in my bibliography deal with the surgery of the heart itself or with the surgery of wounds of pericardium.

Loison on wounds of the heart and pericardium, in an exhaustive paper in Revue de Chir., 1899, vol. 19, gives in

his table four cases of pericardotomy for wounds of pericardium, viz., cases of Cappelen, Eiselberg, Garber and Reidel. Two of these, Eiselberg's and Garber's cases, are cases of operation for suppuration following wounds.

A new method of opening the chest in surgery of the heart and pericardium is advanced by Wehr. (Archiv. f. Klin. Chir., band 59, p. 949.) He recommends it especially where large exposure is necessary in wounds of heart itself. It consists of making an elliptical flap of skin, bone and cartilage, cutting across sternum at the base of fourth cartilage and again at the xiphoid joint, and by a curving sweep taking in fourth, fifth, sixth and seventh costal cartilage, turning this flap back with the right edge of the sternum as a hinge. He does not say he has done this on the living. His method can best be shown by reproducing his cuts, two in number.

Manges, of N. Y., in 1900, reported on cases of pericarditis following pneumonia at the Mt. Sinai Hospital (Med. News, Jan. 20th, 1900) of 500 cases of pneumonia pericarditis developed in only eleven, and of the eleven five died. He thinks all cases of pericarditis as a complication of pneumonia are due to the pneumococcus, and further states that infection with this organism is probable in many cases of so-called idiopathic pericarditis. This same organism finds its way from the pulmonary passages. In the pericardium as in the meninges, the pneumococcus is always a pus-producing organism. It is probable, he says, that many cases of pericarditis in pneumonia are overlooked because the conditions around the heart when the left lung is affected are such as to obscure the most important sign, viz., increase of heart dullness; the to-and-fro murmur of the pericardial friction may last but a few hours.

Connor and Stimson of New York, in 1900 (Med. News, Jan. 20th, 1900) report a case of purulent pericarditis after a severe pneumonia. Operation under local anæsthesia

by Dr. Stimson. The right pleura was cut accidentally; death in a few days from purulent pneumonia.

Ljunggren in his paper (loc. cit.) after reporting his case, the history of which has been translated and put in my list of cases, goes on to say that he has found some new cases which are not in previous lists. Three of these cases are inaccessible, however, and are given thus by him with no details; he merely gives the references.

(Hirschsprung cited by Heyde.

Heyde in a Dissert, auf Kiel, 1896.

Perls in a Dissert, auf Strassburg, 1896.)

The other cases are those of Brentano, which I have given.

It has been possible for the writer to add fourteen cases to the former collections of my own and Roberts, without counting the three cases cited above by Ljunggren, the details of which are not to be had. These make a total of fifty-one cases. (Of this series of cases the annexed tabular view has been made.)

An analysis of the fifty-one cases gives the following facts: Forty-six were for purulent pericarditis, septic; two were for sero-fibrinous pericarditis, rheumatic; one was for hemorrhagic pericarditis, traumatic; two were for serous pericarditis.

The etiology is as follows:—

| | |
|---|---|
| Pneumonia, with and without empyema | 14 |
| Broncho-pneumonia (in a child) | 1 |
| Osteomyelitis | 5 |
| Wounds, gunshot or stab | 4 |
| Blow on chest | 1 |
| Periostitis | 1 |
| Necrosis of nasal bones | 1 |
| Septic throat | 1 |
| Septic arthritis of knee | 1 |
| Abscess of buttock | 1 |
| Empyema without pneumonia | 5 |
| Pleurisy of doubtful origin | 2 |

| | |
|---|---|
| Pleurisy with typhoid fever | 1 |
| Pleurisy with bronchitis | 1 |
| Influenza | 1 |
| Tuberculosis? | 2 |
| Unknown origin | 7 |
| Rheumatic fever and endocarditis more or less acute | 3 |

Of the total fifty-one cases of incision for pericarditis, twenty recovered and thirty-one died, a mortality for all cases of 60.5 per cent.

Of the two operations for simple serous pericarditis both recovered.

Of the fourteen cases in which acute pneumonia, either lobar, bronchial or septic, was the cause of the purulent pericarditis, only four recovered (cases of Bohm, Lillienthal, Porter and Bjorkmann); in two of these the pneumococcus was grown from the pus. The other two do not report bacteriological studies.

Twenty cases were not tapped previous to operation. Local anæsthesia with cocain or eucain was used in six cases.

## CONCLUSIONS.

1. Pericardotomy is indicated in all cases of suppurative pericarditis.

2. Because of the uncertain and varying relations of the pleura and because of the anterior position of the heart, whenever the pericardial sac is distended by fluid, aspiration of the pericardium is a more dangerous procedure than open incision, when done by skilled hands.

3. Incisions of the pericardium can be done quickly and safely by resection of the fifth costal cartilage and in many cases under local anæsthesia.

4. In many cases of serous effusion open incision without puncture will offer less risk and speedier cure than aspiration.

5. The method and detailed technique of the writer proposed in 1897 have been followed out by the majority of recent operators.

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### CASES OF SUPPURATIVE PERICARDITIS TREATED BY INCISION.

CASE 1.—(Hilsmann: "Ueber die Paracentese des Pericardiums," Keil, 1875, Inaug Dissert). Date of operation, 1844. Male, aged twenty-five years. Ill eight months. No complication. Not tapped previously. Incision fourth interspace, a finger's breadth from left edge of sternum. Four tumblerfuls of pus evacuated; much more escaped within a few hours after operation. No irrigation of pericardium. Permanent drainage, wound kept open by inserting probe. After treatment, nearly three months. Recovered. The pericardium was opened behind the sternum though the external wound was a short distance to left of sternum. As the sac was gradually drained, its walls contracted and the pericardial opening moved to the left. In a few days the opening was one and one-half inches to left of sternum and over the fourth rib. In order to empty the sac thoroughly when the external wound was dressed the patient had to bend over as if he was about to stand on his head.

CASE 2.—Langenbeck, Vorlesungen über Chirurgie. Berlin, 1888, p. 449). Date of operation, 1850. Gunshot wound and necrosis of ribs. Not tapped previously. Incision where fifth rib was destroyed. No irrigation. Recovered. Patient had been wounded in a duel in which the bullet shattered five ribs. Necrosis had subsequently occurred, and purulent pericarditis resulted.

CASE 3.—(Rosenstein, Berlin, Klin. Wochenschrift, Jan. 31, 1881, p. 62). Date of operation, 1879. Male, aged ten years. Ill twenty-eight days. Pleuritis a complication. Tapped twice a few days previously with aspirator; pleura also tapped. Incision fourth interspace near sternum. Great quantity of pus evacuated. No irrigation. Permanent drainage with two drainage tubes. After treatment about two months. Recovered. Pleuritis apparently occurred after tapping and incision of pericardium.

CASE 4. — (West. Medico-Chirurgical Transactions, 1883, p. 235). Date of operation, 1882. Male, aged sixteen years. Ill one month. No complication. Tapped three days previously. Incision, fifth interspace, in line of left nipple. Two quarts of pus evacuated. Irrigation, carbolic acid solution, 1-40. Permanent drainage with drainage tube. After treatment, five months. Recovered. Rod shaped bacteria found in pus. Patient in good health nine years afterward. West believes that pericardial adhesions had occurred, but there was no evidence of this condition.

CASE 5. — (Partzevsky, London Medical Record, Feb. 15, 1883, p. 33). Date of operation, 1882? Male, aged twenty-three years. Ill thirteen weeks. Pleuritis a complication. Tapped twice. Incision, fourth interspace. Irrigation salicylic acid solution. Permanent drainage, with drainage tube. After treatment, thirty hours. Death. Autopsy showed cardiac hypertrophy with fatty degeneration, pleural adhesions and pulmonary œdema.

CASE 6. — (West, British Med. Journal, Dec. 8, 1883, p. 1129, and Feb. 21, 1891, p. 404). Date of operation, 1883. Male, aged fourteen years. Pleuritis, pneumonia, abscess of thigh, arthritis were complications. Pericardium opened with bistoury, from wound made to evacuate a supposed pleural effusion. Twenty-four fluid ounces of pus evacuated. After treatment, fourteen days. Death. At autopsy found extensive pericardial adhesions.

CASE 7. — (Savory? reported by Brinton and Collyns, St. Bartholomew's Hospital Reports, 1883, vol. xix., p. 271). Date of operation, 1883. Male, aged nine years. Ill seven weeks. Abscess of shoulder and thigh, pleuritis and pneumonia as complications. Serum obtained from pleura at two previous tappings. Incision fifth interspace in anterior line of axilla through pleural cavity, which was opened because effusion believed to be pleural. Twenty-four fluid ounces of pus evacuated. Irrigation, Condy's fluid. Permanent drainage with drainage tube. After-treatment, fifteen days. Death. Case was one of pyemia. Autopsy showed no communication between left pleura and the track leading through it to pericardium, because adhesions had occurred.

Case 8.—(Scott, New Zealand Medical Journal, July, 1891, p. 268). Date of operation, 1883. Male, aged six years. Ill five weeks. No complication. Tapped with trocar one day previously; irrigation with carbolic acid solution, one per cent. Permanent drainage; two openings an inch apart with drainage tube through both. Recovering.

Case 9.—(Newman (or Sterling), Australian Med. Journal, July 15, 1885, p. 303). Date of operation, 1885. Male, aged thirty-two years. Ill two months. Typhoid fever and pleuritis were complications. Aspiration, five times, fifth time, silver canula allowed to remain for drainage; at fifth tapping carbolic acid solution and tincture of iodine solution used for irrigation; first aspiration one month previously. Incision, third left interspace, incision made where fistula existed from canula, which had been draining the sac for three days. One hundred and seventy-four fluid ounces of pus had been evacuated by five previous tappings. Irrigation with tincture of iodine 1-10 (B. Pharm.). Permanent drainage with rubber tube. After treatment, five days. Death. First aspiration was done in sixth left interspace; second, in fourth right interspace; in third and fourth aspirations no information as to point is given; fifth tapping done in third left interspace. At autopsy a sinus found in second interspace leading from pericardium to the subcutaneous tissue.

Case 10.—(Mikhailov, Med. Oboz. Moscow, 1885, 23, p. 475; Annals of Surgery, November, 1885, quoted from London Med. Record, Aug. 15, 1885). Date of operation, 1885? Female, aged thirty-five years. Ill about three weeks. Pleural, bronchial, and renal lesions as complications. Tapped two days previously. Incision, fourth interspace, near sternum. Two fluid pounds(?) of pus evacuated. Irrigation with boric acid solution. Permanent drainage with drainage tube. After treatment, eighteen hours. Death. Autopsy showed cardiac dilatation with fatty degeneration, pleural and bronchial disease and kidney lesions. Bacteriological examinations of heart and pericardium were negative.

Case 11.—(Gussenbauer. Wiener Med. Wochenschrift. Nov. 22, 1884, p. 1403). Date of operation, 1885(?).

Male, aged thirteen years. Acute osteomyelitis of left shoulder as complication. Fifth rib resected, pericardium opened through pleural cavity because effusion was supposed to be pleural. Irrigation with thymol solution. Permanent drainage by stitching pericardium to edges of wound. Recovered. Small fistule remained when case was reported.

Case 12.—(Rouse (Dickinson's patient) Trans. Clin. Society, London, 1889, p. 48). Date of operation, 1887. Male, aged ten years. Ill about fifteen weeks. Gluteal abscess, pleuritis, abscess of finger as complications. Pleura tapped twelve times; serum obtained. Tapped pericardium three times; last time one week previously. Incision, fifth interspace, right side, close to sternum. No irrigation. Permanent drainage with drainage tube. After treatment, two and one-half months. Recovered. Case considered one of pyæmia. Patient lay on face at times to encourage drainage.

Case 13.—(Underhill, Edinburgh Hospital Reports, 1896, 4, p. 200). Date of operation, 1887. Female, aged six years. Ill two days. Pyemia; had periosteal abscess of tibia a few weeks previously; autopsy showed abscesses in kidneys and pleura. Aspirated twice, last time five days before incision. Incision, fifth left interspace, close to border of sternum. Over ten fluid-ounces of pus evacuated. No irrigation. Permanent drainage with drainage tube. After treatment, five days. Death. Several hemorrhages from interior of pericardium. Autopsy showed that the bleeding probably came from granulation tissue on inner surface of pericardium, probably the seat of septic emboli.

Case 14.—(Parker, Trans. Clin. Society, London, 1889, vol. 22, p. 60). Date of operation, 1888. Female, aged nine years. Ill six and one-half weeks. Osteomyelitis of tibia and suppurative arthritis of knee as complications. Tapped four days previously. Incision, fourth interspace along left border of sternum, with resection of one inch of fifth costal cartilage. A large quantity of pus and lymph evacuated. Irrigated. Permanent drainage, pericardium stitched to edges of wound. Death occurred during irrigation, immediately after incision. Pus

did not flow well; it was thick and contained membraniform shreds; hence irrigation was adopted. Operator believed death was caused by irrigating fluid collecting in the pericardium, as the opening in the pericardium became plugged with lymph. Pressure on heart proved fatal. Patient was pyemic. No special lesion found in chest at autopsy, except pericardial changes.

Case 15.—(Halsted, University Med. Magazine, vol. 6, p. 248). Date of operation, 1890. Male, aged thirty-six years. Ill over three weeks. Acute necrosis of bones of nose, albuminuria, and congestion of right lung as complications. Not tapped previously. Incision, fourth interspace midway between nipple and sternum. Over a quart of pus evacuated. No irrigation. Permanent drainage with gauze plug. After treatment, seventeen days. Death. Great improvement after operation. Dr. Osler, who reports case, calls the pericarditis septic, and attributes the fatal result to a probable myocarditis. No autopsy was made.

Case 16.—(Delorme, Revue de Chirurgie, 1895, vol. 15, p. 1008). Date of operation, 1890. Male, ill eighteen days. Double pleuritis as complication. Empyema operation. Not tapped previously. Incision fourth interspace, a little outside of internal mammary vessels. A few drops of pus evacuated. No irrigation. After treatment, a few moments. Death. Patient died of asphyxia, which had caused the pericardial operation to be hurriedly undertaken. Autopsy showed front of heart adherent to pericardium, and about 500 grammes of sero-pus collected at the base and sides of the heart and a great quantity of false membranes that could only have been removed by a large opening, followed by washing and direct extraction. The opening in the fourth space was over the adherent region.

Case 17.—(Davidson, British Med. Journal, March 14, 1891, p. 578). Date of operation 1890. Male, aged six years. Ill over four weeks. Metatarsal necrosis, subperiosteal abscess at eighth rib on right side; empyema, pneumonia as complications. Not tapped previously. Incision, at fifth interspace. Eight fluid ounces evacuated. No irrigation. Permanent drainage with drainage tube.

After treatment, seven days. Death. Autopsy showed no pericardial adhesions; pneumonia. Case was considered septic.

Case 18.—(Davidson, Brit. Med. Journal, March 14, 1891, p. 578). Date of operation, 1890. Male, aged six and three-fourths years. Ill nearly four weeks. Empyema as complication, which had been operated upon. Pericardium not tapped. Incision, fourth interspace, one inch from left edge of sternum. Several fluid ounces of pus evacuated. No irrigation. Permanent drainage with drainage tube. After treatment, over seven weeks. Recovered.

Case 19.—(Teale (Bronner's patient) British Med. Journal, February 14, 1891, p. 350). Date of operation, 1890. Female, aged eleven years. Ill thirty-four days. Influenza, pneumonia, empyema as complications. Empyema operated on previously. Incision without aspiration through fourth interspace one inch from left border of sternum. Nearly two pints of pus evacuated. Irrigation, iodoform and glycerin, solution of boric acid and carbolic acid. Permanent drainage with drainage tube. After treatment, twenty-six days. Death. No autopsy made, but a probe passed into pericardium discovered no adhesions, though soft granulations were felt toward base of heart.

Case 20.—(Deaver, University Med. Magazine, 1894, Vol. 6, p. 297). Date of operation, 1890. Male, aged twenty-one years. Ill eighteen days. Synovitis? of knee as complication. Aspiration one day previously. Incision, fifth interspace, three inches from middle line. Not much pus evacuated, but seventeen fluid ounces removed by aspiration the day before. No irrigation. Permanent drainage with rubber tube. After treatment, thirteen days. Death. Autopsy showed much fibrinous exudate within pericardium.

Case 21.—(Sievers, Zeitsch. für Klin. Med., 1893, 23. p. 26). Date of operation, 1892. Female, aged twenty-two years. Ill five weeks. Pleuro-pneumonia, empyema on both sides, nephritis as complications. Tapped seven days previously with trocar. Incision, third interspace, two centimetres to left of sternum. Great quantity of

pus evacuated. No irrigation. Permanent drainage. After treatment, eight days. Death. Found bacilli in fluid. Autopsy showed acute nephritis, pleuro-pneumonia, pericardial adhesions and some change in heart muscle. Pericarditis was of a septico-pyemic nature. Operator chose third interspace because fourth and fifth interspaces were very narrow.

Case 22.—(Körte, Verhandl d Berl. Med. Gesellsch (1892) 1893, 23, p. 2). Date of operation, 1891. Female, aged seven years. Osteomyelitis of both tibiæ, as complication. Tapped previously. Incision with resection of five centimetres of fifth rib and cartilage. Evacuated half a litre of pus. Irrigation, lysol solution, 1-2 per cent. Permanent drainage not mentioned. After treatment, twelve days. Death. Autopsy showed numerous foci of pus in fissures of cardiac muscle, in papillary muscles of mitral valve, and in kidneys. Caseous mass in lung and evidence of pleuritis were found. Pus evacaated contained staphylococci, streptococci and bacilli.

Case 23.—(Eiselberg, Weiner Klin. Wochenscrift, January 10, 1895, p. 21). Date of operation, 1894. Male, aged seventeen years. Ill over four and one-half months. Wound made by knife in region of heart, which had healed, left pleuritis and pneumonia followed third tapping. Tapped three times previously. Resection of fourth cartilage. Two litres of pus evacuated. Irrigation, solution of salicylic acid; then iodoform and glycerin. Permanent drainage, two tubes. After treatment, about six weeks. Recovered. Drains kept in pericardium seventeen days.

Case 24.—(Edwards, Trans. Med. Soc. of State of California, 1893, p. 166). Date of operation, 1892 (?) Female, aged six years. Succulated empyema, operation with resection of ribs; mediastinitis, nephritis as complications. Not tapped previously. Incision, one fourth inch within and above position of apex beat. Nine fluid ounces of pus evacuated. No irrigation. Permanent drainage with rubber tube. Death. Began as mediastino-pericarditis, with secondary pleurisy; at least this was the belief of Edwards, after an autopsy.

CASE 25.—(Jacobson, Operations of Surgery, London, 1897, p. 590). Female, aged fourteen. Œdema of lungs as complication. Incision, fifth interspace, right side, a little outside of sternum. Forty-six fluid ounces of pus evacuated. Death occurred from œdema of lungs.

CASE 26.—(Gabszewiez, Gaz. Leklarsk. Warsaw, 1892, 2 Ser. 12 p. 1070). Date of operation, 1892. Male, aged twenty-two years. Ill about one month. No complications. Not tapped previously. Resection of fifth costal cartilage. Large quantity of pus evacuated. Irrigation, boric acid solution. Permanent drainage by iodoform-gauze for eighteen days. Recovered.

CASE 27.—(Robinson, Lancet, November 21, 1896, p. 1460). Date of operation, 1893. Male, aged sixteen years. Ill twenty days. Sore throat and swelling of left wrist were first symptoms. Aspiration one day previous. Resection of sixth rib. Two quarts of pus evacuated. No irrigation. Permanent drainage with one drainage tube. After treatment, about two months. Recovered.

CASE 28.—(Marsh, Lancet, November 21, 1896, p. 1460). Male, aged fourteen years. Incision below nipple. No permanent drainage. After treatment, four days. Death.

CASE 29.—(Klefberg, Tidskrift i Militär, Helsovord 17, Arg. 1892, Stockholm, from Shattuck and Porter, Boston Med. and Surg. Journal, May 6, 1897, p. 444). Male, aged nineteen years. Influenza previously. Aspiration previously and withdrawal of one hundred and fifty grammes of pus. Incision in fourth interspace. One litre of pus evacuated. After treatment, six days. Death. Autopsy showed pus in right ankle and sterno-clavicular joints.

CASE 30.—(Bohm, Deutsche medicin Wochenschrift, Nov. 26, 1896, p. 769). Date of operation, 1894. Male, aged thirty-three years. Ill one month. Influenza previously. Pleuro-pneumonia as complication. Tapped pericardium three times. Incision in third interspace. One litre of pus evacuated. Irrigated with boric acid solution. Permanent drainage with gauze. After treatment, three months. Recovered. Schleich's infiltration method of local anæsthesia was used. Wound closed in three weeks. Out of bed in four and one-half weeks. Well a year later.

CASE 31. — (Allen, personal communication from Dr. D. P. Allen, Cleveland, Ohio, to Dr. Roberts). Date of operation, 1892. Male, aged eleven years. Empyema, requiring costal resection one year before. Aspiration of pericardium previously. Resection of sixth rib below and inside of left nipple. Two quarts of pus evacuated. Irrigation with sterile water. Permanent drainage with gauze. After treatment, twenty days. Death.

CASE 32.—(Stoker, O'Carroll's patient, Dublin Journal of Med. Science, July, 1896, p. 11). Date of operation, 1892. Male, aged twenty years. Ill thirty-four days. Pneumonia. Left pleural effusion subsequent to operation. Aspiration or pericardium five days previously. Incision in fourth interspace midway between nipple and sternum. No irrigation. Permanent drainage on third day by tube. After treatment, thirty days. Death. Fränkel's diplococcus found in pus.

CASE 33.—(Björkman or Hackzell, Hygeia, Stockholm, 1896, lviii., pt. 2, 189). Date of operation, 1895(?). Female, aged twelve years. Ill about three and one half weeks. Had influenza and pleuro-pneumonia previously. Aspirated previously in fourth interspace. Resection, fifth and sixth ribs. Evacuated from 400 to 500 c.c. of pus. Irrigation with boric acid solution. Permanent drainage with two drainage tubes. Duration of treatment six weeks. Recovered. Pleural cavity was opened and some serum evacuated by resection. The pleural opening was then closed with sutures.

CASE 34.—(Porter, Shattuck's patient, Boston Med. & Surg. Journal, May 6, 1897, p. 438). Date of operation, 1895. Male, aged twenty-six years. Ill thirty-three days. Pneumonia before pericarditis. Empyema after pericardotomy, requiring resection. Aspiration two days previously. Incision in fifth interspace one and one-half inches to left of sternum. One quart of pus evacuated. Irrigation with sterile salt solution. Permanent drainage with two rubber tubes. Recovered. Pneumococcus found in pus. Pneumo-thorax and empyema occurred after pericardotomy. Pneumococcus in pus from pleural sac. Erysipelas of back and shoulder occurred. On thirty-sixth day wound entirely healed. Empyema sinus finally closed in about ten months.

CASE 35.—(Garber, Journal American Med. Association, June 26th, 1897, p. 1223). Date of operation, 1897. Female, aged twenty-one years. Ill two weeks. Pericarditis, caused by penetrating wound with steel crochet needle. Not tapped previously. Incision in fifth interspace, one and one-half inches to the left of the sternum. Evacuated about one fluid-ounce of thick pus and also some puro-sanguinolent fluid. Irrigation with salt solution. Permanent drainage with gauze. After treatment, twenty-four days. Recovered.

BRENTANO'S CASES.

CASE 36.—(Brentano, Dent. Med. Work, 1898, 506). Child, 7 years old. Complication of acute osteomyelitis. Symptoms of effusion on sixth day. Ten days later, pericardotomy after diagnostic puncture with Pravaz syringe, resection fifth cartilage. Much pus. Pyemia. Death in twelve days.

CASE 37. — Brentano (ibid). Child, seven years old. Osteomyelitis. Practically the same as above. The operation in these two children was followed immediately by improvement in pulse and general condition, but within a few days (twelve), as shown by post-mortem, pyemia caused death. No cultures from pus reported. At post-mortem the following unusual condition was found. Abscesses, multiple in the muscle of the heart itself in both cases, one of which in second case had perforated into the left ventricle by extensive involvement of the walls of ventricle and caused sudden death by great hemorrhage into the drained pericardium.

CASE 37.—Brentano (ibid). Male, 15 years old. Rheumatic endocarditis and subacute sero-fibrinous pericarditis. Resection of the fifth cartilage after incision, presumably in fifth space without resection, had injured pleura. Pericardium very thick, fibrinous adhesions to wall of the heart. Sero-fibrinous fluid. No cultures. Death in three days with practically no improvement in symptoms.

CASE 39.—Brentano (ibid). Male, thirty-one. Practically same as above save that resection of rib cartilage was at once begun.

In these cases he thinks operation was a mistake because of the chronic cardiac and rheumatic condition. It does not

appear from his article what symptoms and signs caused him to operate.

In these two cases so adherent was heart and pericardium that puncture, he says, could hardly have failed to have done serious mischief.

Case 40.—Brentano (ibid). A case of serous pericarditis following rheumatic endocartitis in a girl aged ten. Very sick case from large exudation. No puncture, but incision after resection of the fifth cartilage. 300 c.c. of sero-sanguinolent fluid removed. No cultures reported. Immediate and marked improvement. Drainage with iodoform gauze. Fluid, clear, flowed from the wound for a day or two, but in eight days only a fine granulating dry sinus was left. Recovery.

Case 41.—Lilienthal's case. Reported before N. Y. Acad. of Med., Nov. 13th, 1899 (Med. News, Nov. 25, 1899). Male, aged fifteen. Trilobar pneumonia with very high fever, pneumococci in sputum and later streptococci. Six weeks after very sick, pericardial friction sounds and dullness, aspirated and eighteen ounces of pus, giving pure culture pneumococcus, withdrawn. Then under eucain local anæsthesia, incision, with resection of fourth and fifth costal cartilages. Pericardial sac much thickened, pleura not opened. Forty ounces of pus. Irrigation with salt solution. Wound left open, no drainage tubes or gauze. Recovery. Adhesion of pericardium and the heart with the scar was evident after recovery, by the retraction of the tissues at each systole.

Case 42.—Case of Allingham & Ogle. (Lancet, March 10, 1900.) Male, age 26(?). Pericardial infection from small collection of pus in pleural cavity to right of pericardium. Chronic disease of lungs. No acute sepsis. Symptoms of pericardial effusion baffling. Incision and resection of fifth rib cartilage. Pleura not opened. Pericardium and pleuræ firmly adherent to chest wall. Sixteen ounces of non-offensive pus evacuated. No cultures. Great masses of fibrin in sac and adhering to sac and to heart. As much of this as possible removed with fingers and sponges; about five ounces back of heart could not be reached. Sac stitched to skin. Irrigation with hot water. Drainage not mentioned, but wound was left open. Temporary im-

provement. Death in fourteen hours. Autopsy revealed cause of infection.

Case 43. — Robinson's second case. — (British Med. Journal, 1898, Nov. 26). Boy, aged 4. Broncho-pneumonia for three and one-half weeks. Operated when nearly moribund. Kind of anæsthesia not given. Incision over fifth cartilage, one inch removed. Pleura not opened. Ten ounces of pus and sero-turbid fluid. Cultures showed pure cultures of pneumococcus. Fibrin on anterior wall pericardium. No irrigation. Gauze drainage. Marked improvement after operation for twenty-four hours. Death on third day after operation. No post-mortem.

Case 44.—Sevestre's case.—(Lancet, April 23, 1898). Male, 22. Acute pneumonia and pneumococcus pericarditis. Very sick case. Aspiration in fourth space close to left border of the sternum, twenty-three ounces of pus giving pure cultures pneumococcus. Under cocain the next day as preliminary to operation aspirating needle put in the same place with result of getting only bright blood. Three days after the operation under cocain(?) in fourth space. No mention of pleura made. No resection. Forty-five ounces of pus evacuated. Sac washed out and drainage tube put in. Three days after this double empyema and resection of a rib on both sides under chloroform (at intervals of two days) pneumococci in pleural pus. Death, eighteen days after first pericardial aspiration.—(Probable infection of pleural cavities at first operation.)

Case 45.—C. Mansell-Moulin's case.—(Trans. Clin. Soc., vol. 30, p. 217, 1897). Male, 28. Blow over chest while playing foot-ball. Collapse soon after followed by cardiac dulness next day with cough and pain on breathing. Continued for nearly a month with less pain and easier breathing except that cardiac dulness increased spreading to right of sternum. About a month after injury sudden great dyspnœa and pain. Operation under cocain anæsthesia. Incision through fifth space. Nothing said about resection. Pleura opened. About six pints thin dark bloody fluid which clotted very rapidly was removed. Drainage tube left in for twenty-four hours. Good recovery though left lung remained collapsed for very long time. No cultures mentioned.

CASE 46.—Conners' and Stimson's case.—(Med. News, Jan. 20, 1900). Sex and age unknown. Severe pneumonia. Operation under local anæsthesia. Pleura (right opened) incision not given. Death in a few days.

CASE 47.—Case of Bexman. Incision in second space for purulent pericarditis. Recovery. Mentioned by Voinitsch-Siansjensky elsewhere. (See Tables.)

CASE 48.—Case of Ljunggren.—(Translated from history of the case in Nordisk. Med. Arkiv., Stockholm, 1898, Band ix., Heft 6, New Series). Male, laborer, aged 24. Well previously save that some years before had disease in his left lung, the nature of which he did not know. On Oct. 28, 1896, had a severe chill with fever, also shortness of breath and coughing, and pain in the left breast in front. Could not get up. Could not lie on the left side without very difficult breathing. Cough and fever continued till he was admitted to hospital the 17th of November.

On entering hospital this condition was noted: The left half of the chest is more prominent than the right with dulness on percussion over the whole left front from second rib down, this dulness reached to about 1 c.m. to the right of the right sternal border and over the entire lower half of the thorax to the back and as high as the top of the scapula. Respiratory sounds could not be heard over any of this dull area. Heart beats could not be felt. Heart sounds could be heard, though faintly. Distension of abdomen, especially epigastric. Liver dulness lost. Breathing of the costal type and frequent. The face was cyanosed and anxious. There was some œdema of the feet. Temperature was 39.5 C. Pulse even and weak, 110. Pain over the left chest and difficulty in breathing complained of by patient. Under expectant treatment he became worse each day. Tapped twice in the back for supposed empyema without result, no fluid found. As the fever lessened, breathing became more difficult and heart's action weaker. Aspiration (diagnostic) with hypodermic needle in the fifth intercostal space a little below and inside of the left nipple where fluctuation was felt. Under a small amount of chloroform an incision in fifth space beginning 2 c.m. inside and below nipple and ending 6 c.m. outside.

| No. of Case. | Operator. | References and Date. | Etiology. | Incision and Technique. | Irrigation, Drainage. | Result. | Remarks and Bacteriology. |
|---|---|---|---|---|---|---|---|
| 1 Male 23 yrs. | Hillsmann | Inaug. Dissert Kiel, 1875. Date operation, 1844. | Not given. | In 4th space near sternum. Pericardium opened without first aspirating. | No irrigation. Probing wound daily. | Recovered in 3 months. | |
| 2 ? | Langenbeck | Vorlesungen über Chirurgie p. 449 Berlin, 1888. | Gunshot wound and necrosis rib, (5th). | Incision through lost rib substance. | No irrigation. Drainage? | Recovered. | |
| 3 Male 10 yrs. | Rosenstein | Berlin K. Woch. Jan. 31, 1881. Date operation, 1879. | Pleurisy? As to primary or secondary. Etiology doubtful. | Tapped once. Incision in 4th space near sternum. | No irrigation. Drained with two rubber tubes. | Recovered in 2 months. | Very large quantity of pus in the sac. |
| 4 Male 16 yrs. | West | Med. Chir. Trans. 1883, p. 235 | Etiology? Perhaps tubercular. No complications. | Tapped first. Incision in 5th space, line of nipple. | Irrigated with carbolic 1 to 40. Tube drainage. | Recovered in 5 months. | 2 quarts of pus; Rod shaped bacteria in it. ?Tubercle bacilli. |
| 5 Male 23 yrs. | Partzevsky | London Med. Rec., 1883, Feb. 15. | ? Pleurisy. | Tapped twice, then cut in 4th space. | Irrigated with sal. acid. Double tube drainage. | Death in 30 hours. | |
| 6 Male 14 yrs. | West | Brit. Med. Jour., 1883, Dec. 8th, and 1891, Feb. 21. | Pneumonia and general sepsis. | Opened with bistoury in operation for pleural effusion. Site? | ? | Death in 14 days. | Cause of death; general sepsis. |
| 7 Male 9 yrs. | Savory and Brinton and Collyns | St. Barthol. Hosp. Rep., 1883, Vol. XIX., 279. | Septic. Pneumonia or pleurisy with multiple abscesses. | Incision 5th space. Pleura opened. | Irrigation with Condy's fluid. Tube drainage. | Death in 15 days. | Cause of death; Pyemia. Incision in ant. axillary line for the empyema primarily. |
| 8 Male 6 yrs. | Scott | New Zealand Med. Jour., 1891, July, p. 268. | Unknown. No complications. | Tapped once. Cut in 5th space near apex heart. Pleura not mentioned. | Irrigation with 1½ per c. carbolic. 2 tubes drained. | Recovered, time? | Two incisions 1 in. apart, drain-tube in each. |
| 9 Male 32 yrs. | Newman, or Stirling | Austral. Med. Jour., 1885, July 15. | Typhoid fever and Pleurisy. | Aspirated 5 times. Cut 3d space in old trocar track. | Irrigation with iodine, 1 to 10. Tube drained. | Death in 5 days. | 174 oz. pus removed by 5 tappings. |
| 10 Female 35 yrs. | Mikhailov | Lond. Med. Rec., 1885, Aug. 15. | Bronchitis and pleural lesion? | Tapped previously. Cut in 4th space near sternum. Pleura not mentioned. | Boric acid. Tube. | Death in 18 hours. | Bacteriology exam. of sac.? at autopsy was negative. |
| 11 Male 13 yrs. | Gussenbauer | Wien. Med. Woch. 1884, Nov. 22. | Osteomyelitis acute. | Through resection 5th rib. Pleura opened. | Thymol sol. Sac stitched up. | Recovered time? | |
| 12 Male 10 yrs. | Rouse, and Dickinson | Trans. Clin. Soc., 1889, p. 48. | Abscesses buttock and fingers, with pleurisy. | Tapped 3 times. Cut in 5th space on right. Pleura not mentioned. | No irrigation. Tube drainage. | Recovered in 2½ months. | |
| 13 Female 6 yrs. | Underhill | Edinb. Hosp. Reps., 1890, p. 200. | Pyemia from periostitis tibiæ. | Tapped twice. Cut in 5th space near sternum. | No irrigation. Tube drainage. | Death in 5 days. | Cause death; pyemia and hemorrhages from interior of pericardium. |
| 14 Female 9 yrs. | Parker | Trans. Clin. Soc., London, 1889, Vol. XXII., p. 60. | Osteomyelitis and arthritis. | Tapped. Incision with resection 5th cartilage. Pleura not opened. | Irrigation. Death during this. | Death on the table. | Cause of death said to have been poor return of irrigating fluid and pressure on heart. |
| 15 Male 30 yrs. | Halstead | Univ. Med. Mag., Vol. VI., p. 248. Date operation, 1890. | Necrosis of nasal bones. | Incision, 4th space half way between nipple and sternum. Pleura not mentioned. | No irrigation. Gauze drain. | Death in 17 days. Pyemia. | |
| 16 Male ? | Delorme | Revue de Chirurgie, 1895, Vol. XV., p. 1008. | Empyema. | Incision 4th space near sternum. Only a few drops pus. | No irrigation or drainage. | Death in few minutes. | Autopsy showed that ant. wall of heart was adherent and about a pint of pus was behind and at the sides. |
| 17 Male 6 yrs. | Davidson | Brit. Med. Jour., 1891, March 14. | Pyemia and septic pneumonia after necrosis metatarsus. | Incision in 5th space after aspiration. | No irrigation. Tube drain. | Death in 7 days from sepsis. | |
| 18 Male 64 yrs. | Davidson (2d case) | Ibid. | Empyema. | Not tapped first. Cut in 4th space near sternum. | No irrigation. Tube drain. | Recovered in 7 weeks. | |
| 19 Female 11 yrs. | Teale or Brouner | Brit. Med. Jour., Feb. 14, 1891. | Pneumonia and empyema. | Tapped first. Incision in 4th space 1 inch from sternum. | Boric and carb. ac. Iodoform glycerine; tube drain. | Death in 26 days. | No autopsy. |
| 20 Male 21 yrs. | Deaver | Univ. Med. Mag., 1894, Vol. VI., 297. | Synovitis(?) knee, Septic knee probably. | Aspirated first. Incision in 5th space, 3 inches from middle line. | No irrigation. Tube drain. | Death in 13 days. | Autopsy showed much fibrinous exudate in sac. |
| 21 Female 22 yrs. | Sievers | Zeitschr. f. Klin. Med. 1893, XXIII., p. 26. | Pneumonia and empyema (right). | Tapped 7 days earlier. Incision 3d space near sternum. Left pleura opened. | No irrigation. Tube drain? | Death in 8 days. Pyemia. | "Bacilli" (?) in pus. Left empyema after operation. |
| 22 Female 7 yrs. | Körte | Verhdl. d. Berl. Med. Gesellsch, 1893, XXIII., 2. Date operation, 1891. | Osteomyelitis of both tibiæ. | Tapped previously. Resection of 5th rib and cartilage. | Irrigation with lysol ½ p.c. Drainage? | Death in 12 days from sepsis. | Pus showed streptococci, staphylococci and ? bacilli, and autopsy showed abscess in heart wall. |
| 23 Male 17 yrs. | Eiselberg | Wien. Klin. Wochenschr., 1895, Jan. 10. | Stab wound 4½ months previous. | Tapped 3 times. Resection of 4th cartilage. | Salicyl. acid & iodof. glyc. Two tubes for 17 days. | Recovered in 6 weeks. | Early tappings caused pleurisy & pneumonia. Colon bacilli in fluid. |
| 24 Female 6 yrs. | Edwards | Trans. Med. Soc. of California, 1893. | Empyema and operation for same. "Mediastinitis"(?) | Incision ¾ inch within and above apex beat. | No irrigation. Tube drain. | Death. | ? as to priority of pericarditis or empyema. |
| 25 Female 14 yrs. | Jacobson | Operations of Surgery, London, 1897, p. 590. | Unknown. | Incision in right 5th space close to sternum. | ? ? | Death from œdema of the lungs. | |
| 26 Male 22 yrs. | Gabszewics | Roberts' list. Also Gaz. Leklarsk of Warsaw, 1892, 2d series. | Unknown. | Resection of 5th cartilage. | Boric and sol. iodof. Gauze drain for 18 days. | Recovered. | |
| 27 Male 16 yrs. | Robinson | Lancet, 1896, Nov. 21, p. 1400. | Sore throat (?) septic. | Tapped previously. Resection of 6th rib. Pleura (left opened). | No irrigation. Tube drain. | Recovered in 2 months. | Two quarts of pus. No cultures. |
| 28 Male 14 yrs. | Marsh | Ibid. | Unknown, or not given. | Incision below the nipple. | ? No permanent drainage. | Death in 4 days. | |
| 29 Male 19 yrs. | Klefsburg of Stockholm | Dr. Porter's list and Roberts' list. | Influenza. | Tapped previously. Incision in 4th space. Local anæsthesia. | ? ? | Death in 6 days from sepsis general. | Autopsy showed pus in joints. |
| 30 Male 33 yrs. | Rohm | Deut. Med. Wochensch, 1896, Nov. 26. | Influenza and pneumonia. | Tapped 3 times. Incision in 3d space. Local anæsthesia, "Schleich." | Boric acid sol. Gauze drain. | Recovered in 4½ weeks. | |
| 31 Male 11 yrs. | Allen of Cleveland. | Roberts' list. Date operation, 1892. | ? of old empyema. | Tapped previously. Resection of 6th rib below and inside the nipple. | Sterile water. Gauze drain. | Death in 20 days. | |
| 32 Male 20 yrs. | Stoker (O'Carroll's case) | Dublin Jour. Med. Sci., July, 1896. Date operation, 1892. | Pneumonia & pleural effusion. | Tapped previously. Incision in 4th space between upper and sternum. | No irrigation. Tube drain. | Death in 30 days. | Pneumococcus in pus. |
| 33 Female 12 yrs. | Bjorkmann or Hacksell of Stockholm. | Roberts' list. | Influenza and pneumonia. | Tapped previously. Resection 5th & 6th ribs. Pleura opened and sutured. | Boric acid sol. 2 tubes. | Recovered in 6 weeks. | |
| 34 Male 26 yrs. | C. B. Porter | Boston Med. & Surg. Jour., 1896, May 6. Also Tr. Am. S'rg. Assoc., '97. | Pneumonia. | Tapped previously. Incision in 5th space 1½ inches left of sternum. | Salt sol. Two tubes. | Recovered. | Empyema and pneumothorax after operation. Pneumococcus grown from pus from pericarditis & empyema. |
| 35 Female 21 yrs. | Garber | Journal Am. Med. Assoc., 1897, June 26. | Stab wound with needle 2 weeks before. | Incision in 5th space 1½ inches left of sternum. | Salt sol. Gauze drain. | Recovered in 24 days. | Sanguinolent fluid and about an oz. of pus. No cultures. |

The above 35 cases are all given in Roberts' list. In addition, the following 16 cases have been found and tabulated. Two in Dr. Porter's list, Pepper case and Rullier.

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 36 | Peterson tion, 1892. | Cited by Voinitsch— | Unknown. | Incision in 3d space. | ? | Recovered. | |
| 37 Male age? | Riedel | Revue de Chir, 1896-993. Centralbl. f. Chir., 1897, p. 59. | Pistol wound 10 days before. | Resection "several" ribs and opening pericardium. No details given. | ? No drainage. | Death. | Bloody, thin fluid. No cultures. Resection of rib for draining pleura right after wound. For pericardotomy 10 days later. |
| 38 | Ljunggren of Stockholm. | Nord. Med. Arkiv., 1896. U. S. Bd IX f. 6. | Pneumonia acute. | Diagnostic aspiration. Incision in 5th space between sternum & nipple. Later resection 5th rib. | Irrigation with salt sol. 2 tubes. | Recovered. | B. Pyocyaneus in the pus, which was green in color. |
| 39 | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Female 6 yrs. | [illegible] | 1896, p. 200. | ostitis tibiæ. | space near sternum. | No irrigation. Tube drainage. | Death in 5 days. | Cause death; pyemia and hemorrhages from interior of pericardium. |
| 14 Female 9 yrs. | Parker | Trans. Clin. Soc., London, 1889, Vol. XXII., p. 60. | Osteomyelitis and arthritis. | Tapped. Incision with resection 5th cartilage. Pleura not opened. | Irrigation. Death during this. | Death on the table. | Cause of death said to have been poor return of irrigating fluid and pressure on heart. |
| 15 Male 36 yrs. | Halstead | Univ. Med. Mag., Vol. VI., p. 248. Date operation, 1890. | Necrosis of nasal bones. | Incision, 4th space half way between nipple and sternum. Pleura not mentioned. | No irrigation. Gauze drain. | Death in 17 days. Pyemia. | |
| 16 Male ? | Delorme | Revue de Chirurgie, 1895, Vol. XV., p. 1008. | Empyema. | Incision 4th space near sternum. Only a few drops pus. | No irrigation or drainage. | Death in few minutes. | Autopsy showed that ant. wall of heart was adherent and about a pint of pus was behind and at the sides. |
| 17 Male 6 yrs. | Davidson | Brit. Med. Jour., 1891, March 14. | Pyemia and septic pneumonia after necrosis metatarsus. | Incision in 5th space after aspiration. | No irrigation. Tube drain. | Death in 7 days from sepsis. | |
| 18 Male 6½ yrs. | Davidson (2d case) | Ibid. | Empyema. | Not tapped first. Cut in 4th space near sternum. | No irrigation. Tube drain. | Recovered in 7 weeks. | |
| 19 Female 11 yrs. | Teale or Brounar | Brit. Med. Jour., Feb. 14, 1891. | Pneumonia and empyema. | Tapped first. Incision in 4th space 1 inch from sternum. | Boric and carb. ac. Iodoform glycerine; tube drain. | Death in 26 days. | No autopsy. |
| 20 Male 21 yrs. | Deaver | Univ. Med. Mag., 1894, Vol. VI., 297. | Synovitis (?) knee, Septic knee probably. | Aspirated first. Incision in 5th space, 3 inches from middle line. | No irrigation. Tube drain. | Death in 13 days. | Autopsy showed much fibrinous exudate in sac. |
| 21 Female 22 yrs. | Sievers | Zeitschr. f. Klin. Med. 1893, XXIII., p. 26. | Pneumonia and empyema (right). | Tapped 7 days earlier. Incision 3d space near sternum. Left pleura opened. | No irrigation. Tube drain? | Death in 8 days. Pyemia. | "Bacilli" (?) in pus. Left empyema after operation. |
| 22 Female 7 yrs. | Körte | Verhdl. d. Berl. Med. Gesellsch, 1893, XXIII., 2. Date operation, 1891. | Osteomyelitis of both tibiæ. | Tapped previously. Resection of 5th rib and cartilage. | Irrigation with lysol ½ p.c. Drainage? | Death in 12 days from sepsis. | Pus showed streptococci, staphylococci and ? bacilli, and autopsy showed abscess in heart wall. |
| 23 Male 17 yrs. | Eiselberg | Wien. Klin. Wochenschr., 1893, Jan. 10. | Stab wound 4½ months previous. | Tapped 3 times. Resection of 4th cartilage. | Salicyl. acid & iodof. glyc. Two tubes for 17 days. | Recovered in 6 weeks. | Early tappings caused pleurisy & pneumonia. Colon bacilli in fluid. |
| 24 Female 6 yrs. | Edwards | Trans. Med. Soc. of California, 1893. | Empyema and operation for same. | Incision ¼ inch within and above apex beat. | No irrigation. Tube drain. | Death. | ? as to priority of pericarditis or empyema. |
| 25 Female 14 yrs. | Jacobson | Operations of Surgery, London, 1897, p. 590. | "Mediastinitis" (?) Unknown. | Incision in right 5th space close to sternum. | ? ? | Death from œdema of the lungs. | |
| 26 Male 22 yrs. | Gabszewiez | Roberts' list. Also Gaz. Leklarsk of Warsaw, 1892, 2d series. | Unknown. | Resection of 5th cartilage. | Boric and sol. iodof. Gauze drain for 18 days. | Recovered. | |
| 27 Male 16 yrs. | Robinson | Lancet, 1896, Nov. 21, p. 1460. | Sore throat (?) septic. | Tapped previously. Resection of 6th rib. Pleura (left opened). | No irrigation. Tube drain. | Recovered in 2 months. | Two quarts of pus. No cultures. |
| 28 | Marsh | Ibid. | Unknown, or not | Incision below the nipple. | ? | Death in 4 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 14 yrs. | | | | | | lungs. | |
| 26 Male 22 yrs. | Gabszewiez | Roberts' list. Also Gaz. Lekiarsk of Warsaw, 1892, 2d series. | Unknown. | Resection of 5th cartilage. | Boric and sol. iodof. Gauze drain for 18 days. | Recovered. | |
| 27 Male 16 yrs. | Robinson | Lancet, 1896, Nov. 21, p. 1460. | Sore throat (?)septic. | Tapped previously. Resection of 6th rib. Pleura (left opened). | No irrigation. Tube drain. | Recovered in 2 months. | Two quarts of pus. No cultures. |
| 28 Male 14 yrs. | Marsh | Ibid. | Unknown, or not given. | Incision below the nipple. | ? No permanent drainage. | Death in 4 days. | |
| 29 Male 19 yrs. | Klefburg of Stockholm | Dr. Porter's list and Roberts' list. | Influenza. | Tapped previously. Incision in 4th space. Local anæsthesia. | ? ? | Death in 6 days from sepsis general. | Autopsy showed pus in joints. |
| 30 Male 33 yrs. | Bohm | Deut. Med. Wochensch, 1896, Nov. 26. | Influenza and pneumonia. | Tapped 3 times. Incision in 3d space. Local anæsthesia, "Schleich." | Boric acid sol. Gauze drain. | Recovered in 4½ weeks. | |
| 31 Male 11 yrs. | Allen of Cleveland. | Roberts' list. Date operation, 1892. | ? of old empyema. | Tapped previously. Resection of 6th rib below and inside the nipple. | Sterile water. Gauze drain. | Death in 20 days. | |
| 32 Male 20 yrs. | Stoker (O'Carroll's case) | Dublin Jour. Med. Sci., July, 1896. Date operation, 1892. | Pneumonia & pleural effusion. | Tapped previously. Incision in 4th space between upper and sternumn. | No irrigation. Tube drain. | Death in 30 days. | Pneumococcus in pus. |
| 33 Female 12 yrs. | Bjorkmann or Hacks-ll of Stockholm. | Roberts' list. | Influenza and pneumonia. | Tapped previously. Resection 5th & 6th ribs. Pleura opened and sutured. | Boric acid sol. 2 tubes. | Recovered in 6 weeks. | |
| 34 Male 26 yrs. | C. B. Porter | Boston Med. & Surg. Jour., 1896, May 6. Also Tr. Am. S'rg. Assoc.,'97. | Pneumonia. | Tapped previously. Incision in 5th space 1½ inches left of sternum. | Salt sol. Two tubes. | Recovered. | Empyema and pneumothorax after operation. Pneumococcus grown from pus from pericarditis & empyema. |
| 35 Female 21 yrs. | Garber | Journal Am. Med. Assoc., 1897, June 26. | Stab wound with needle 2 weeks before. | Incision in 5th space 1½ inches left of sternum. | Salt sol. Gauze drain. | Recovered in 24 days. | Sanguinolent fluid and about an oz. of pus. No cultures. |

The above 35 cases are all given in Roberts' list. In addition, the following 16 cases have been found and tabulated. Two in Dr. Porter's list, Pepper case and Rullier.

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 36 | Rexman Date of operation, 1892. | Cited by Volnitsch—Sianojensky Revue de Chir, 1898-995. | Unknown. | Incision in 2d space. | ? | Recovered. | |
| 37 Male age? | Riedel | Centralbl. f. Chir., 1897, p. 50. | Pistol wound 10 days before. | Resection "several" ribs and opening pericardium. No details given. | ? No drainage. | Death. | Bloody, thin fluid. No cultures. Resection of rib for draining pleura right after wound. For pericardotomy 10 days later. |
| 38 | Ljunggren of Stockholm. | Nord. Med. Arkiv., 1896. U. S. Bd IX f. 6. | Pneumonia acute. | Diagnostic aspiration. Incision in 5th space between sternum & nipple. Later resection 5th rib. | Irrigation with salt sol. 2 tubes. | Recovered. | B. Pyocyaneus in the pus, which was green in color. |
| 39 Male 15 yrs. | Lillienthal | Med. News, 1899, Nov. 25th. | Double pneumonia. Severe. | Aspirated once. Incision with resection 4th and 5th cartilages with cocain anæsthesia. Pleura not opened. | Salt sol. No drains. Wound left open. | Recovered in 6 weeks. | Pneumococcus in pus in pure culture. Pneumococcus and streptococcus in sputum. |
| 40 Male 26? | Allingham and Ogle | Lancet, March 16, 1900. | Old purulent encysted pleurisy. No acute cause. | Resection 5th cartilage. Pleura not opened. | Hot water. Sac stitched up. No drains. | Death in 14 hours. | 16 oz. pus. No cultures. Much fibrin in sac. |
| 41 Male 4 yrs | Robinson (his 2d case) | Brit. Med. Jour., 1898, Nov. 26. | Broncho-pneumonia. Severe. | Resection 5th cartilage. Pleura not opened. | No irrigation. Gauze drain. | Death in 3 days. | Sero-puriform fluid giving pneumococcus pure. |
| 42 Male 22 yrs. | Sevestre | Lancet, April 23, 1898. | Acute Pneumonia. Severe. | Aspirated first. Then under cocain cut in 4th space. Pleura not mentioned. | Hot water. Tube drain. | Death in 12 days. | Pneumococcus pure. Double empyema with double resection ribs a few days after. |
| 43 Male 28 yrs. | Mansell Moulin | Clin. Soc. Trans., 1897, Vol. 30, 217. | Blow on chest 1 month before. | Under cocain cut in 5th space. Pleura opened. | No irrigation. Tube drain for 24 hours. | Recovery. | Thin bloody fluid. No cultures. Lung collapsed for a long time. |
| 44 ? ? | Conners and Stemson | Med. News, Jan. 20, 1900. | Acute Pneumonia. Severe. | Under cocain, cut on left. Where not given. Right pleura opened. | ? | Death in a few days. | |
| 45 Child 7 yrs. | Brentano | Deut. Med. Woch., 1898, p. 506. | Acute osteomyelitis. | Resection 5th cartilage. Pleura not opened. | Irrigation. Drainage with iod. gauze. | Death in 10 days. | No cultures. Pyemia cause of death. Abscess in heart wall. |
| 46 Child 7 yrs. | Brentano (2d case, like the above) | Ibid. | Acute osteomyelitis. | As above. | As above. | As above. | Profuse hemorrhage from sac from ruptured heart was cause of death. |
| 47 Male 15 yrs. | Brentano | Ibid. | Rheumatism, endocarditis. | Resection 5th cartilage. Pleura injured. | Irrigation. Iod. gauze drain. | Death in 3 days. | Sero-fibrinous fluid with adhesions in sac. No cultures. |
| 48 Male 31 yrs. | Brentano | Ibid. | Rheumatism, endocarditis. | Resection 5th cartilage. Pleura not cut. | Irrigation. Iod gauze drain. | Death. | Sero-fibrinous fluid. No cultures. Heart adherent anteriorly. |
| 49 Female 10 yrs. | Brentano | Ibid. | Rheumatic endocarditis. | Resection 5th cartilage. | Irrigation. Iod. gauze drain. | Recovery in 10 days. | Serous fluid only. No cultures. |
| 50 Male 19 yrs. | Pepper. From Dr. C. B. P. list. | Univ. Med. Mag., 1894. Feb. | Influenza. | Operation first. Small incision and tube in 5th space. | ? | Death in 2 days. | At autopsy little pus, but whole sac distended with fibrin. |
| 51 | Rullier. From Dr. C. B. P. list. | (See Dr. Porter's paper) | ? Serous pericarditis. | Tapped 4 times. Iodine injected in 4th space. | | Recovered. | |

The pleura was adherent. Pericardium opened with great spouting up of pus as result. Collapse of patient, pulse stopped—but compression of left ventricle of heart with operator's fingers caused renewed beating. After this the sac was emptied slowly. Heart was close up to sac wall with a few adhesions between. One and one-half litres of bright green pus were removed. The most of the fluid was localized back and at the sides of the heart. Drainage tubes to back of sac. From pus several cultures were taken, but growths were doubtful. Microscopic examination showed short bacilli that often lay two and two. Cultures from fluid on second day gave B. Pyocyaneus. Steady improvement. On second day considerable fibrin in lumps came from wound. At end of a week after operation no fever and very little difficulty in breathing. At second week after irrigation with salt solution brought away coagula of fibrin. Irrigation about every three days. Because of contraction of sinus through the fifth space operated on again to get more room for irrigation and resected the fifth rib and through this larger opening kept on trying to keep sac clear of fibrin, etc., by irrigation. After this second operation, steady improvement and ultimate recovery. One year after operation normal cardiac dulness and minute fistula which discharged a little very occasionally.

Case 49.—Riedel's case.—(Centralbl. f. Chir., 1897, p. 59). Man, pistol wound. Ten days afterwards resection of several ribs and opening of pericardium. Sanguinolent fluid. Could not drain the sac because of tubes causing irregular pulse. Death. No other details given.

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ARTICLE XIX.

# CLINICAL VARIATIONS IN THE COURSE OF PNEUMONIA ACCORDING TO THE BACTERIOLOGICAL ETIOLOGY.

By FREDERICK H. BAKER, M.D.,
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READ JUNE 12, 1900.

# CLINICAL VARIATIONS IN THE COURSE OF PNEUMONIA ACCORDING TO THE BACTERIOLOGICAL ETIOLOGY.

We know that pneumonia is a septicemia or toxemia, caused by the action of one or more varieties of bacteria upon the lung tissue. We know that it presents a most diversified symptomatology in its onset and throughout its course. Death or recovery may come at the end of a few days or after the lapse of many weeks. The influence of age, personal habits, the physical condition of the patient at the time of the onset, are seen to affect the course of the disease, but the dosage and the nature of the toxins on the one hand, and the bodily resistance on the other, are doubtless the chief factors in the causation of the wide variations as we see them clinically.

Admitting that pneumonia is a bacterial disease, it is hardly satisfying to the clinician to say that a given case of lung infection is a lobar or a lobular pneumonia. The clinician desires, if possible, to go farther, and say this is a pneumococcus infection, or a streptococcus infection, or a mixed infection, etc. Now attempting to do this is not a scientific refinement, a mere question of pathological interest; but it becomes a question not only of diagnosis but of prognosis, and of practical therapeutics, if now or in the future the field of serum therapy is applicable to pneumonia.

In diphtheria and in other membranous diseases of the upper respiratory tract we use cultures to detect which or-

ganism is the cause of the disease. In meningitis we use the fluid obtained by lumbar puncture to diagnose one form of infection from another. In chronic lung disease we examine the sputum to determine the infection. In malaria we examine the blood to determine which variety of the plasmodium is the cause of the disease. In differentiating typhoid fever from diseases closely resembling it in clinical aspects we examine the blood, the urine, and the stools. In differentiating amœbic dysentery from other dysenteries we examine the stools. In differentiating one anemia from another we examine the blood.

Thus it seems to me that while in these diseases just enumerated we may in many cases make an accurate diagnosis from the clinical symptoms,—that is, from the history of the case, the temperature, the pulse, the respiration, auscultation, percussion, and routine examinations, etc., yet in very many we cannot clinch the diagnosis or even make it at all, without calling in the aid of laboratory methods in examining the various fluids, secretions and excretions of the body.

Now as regards the pneumonias,—is there anything in the onset, the appearance, the temperature, the pulse, the respiration, the auscultation or percussion signs, or the course of the disease that will give us helpful or positive symptoms as to what the infection or infections in the lung are? Can we find organisms in the sputum, and if so what deductions can we draw from their presence? Are there any organisms in the blood, or reactions in the blood, or is the urine in any way peculiar to the existing infection?

To begin with, the bacteriology of acute lung infections must be well understood. I shall consider it under the usual clinical divisions of the lobar and the lobular types.

### LOBAR PNEUMONIA.

A review of the published observations of the findings in many hundreds of autopsies of lobar pneumonia made in

different countries show a universal opinion that the pneumococcus is the usual cause of this type of pneumonia. But it is often associated with other organisms. Pearce's analysis of the lobar pneumonia autopsies performed at the Boston City Hospital may be taken as a type of the usual findings. In 110 cases in which the pneumococcus was detected in the pneumonic areas, it was the only organism present in eighty-four cases. In the remaining twenty-six cases it was associated with other organisms,—viz. with the staphylococcus aureus nine times; streptococcus eight times. In the other nine cases with various organisms, chiefly in combination with the pus cocci. His statistics show that the pneumococcus is present constantly in the pleural and pericardial exudates which accompany the pneumonia. In his series of 118 cases the pneumococcus was found in the heart's blood fifty-six times, kidney fifty-one times, spleen forty-seven times, and liver forty-four times. This fact of its getting into the blood current is an important clinical one. Unfortunately we do not know how often it is found in the peripheral blood during life. The other fact, that in over twenty per cent. of the cases it was associated with other organisms, is very significant. How important a rôle the streptococcus or the aureus or their combinations play in causing clinical variations we have no means of determining. It must be borne in mind that probably several other organisms, as the influenza bacillus and Friedlander's bacillus, may cause a true lobar pneumonia.

### LOBULAR PNEUMONIA.

There remains much to be learned concerning the bacteriology of lobular pneumonia, especially that following the acute infectious diseases, except possibly that following diphtheria. An interesting clinical question in the pneumonias following the infectious diseases is whether or not

the pneumonia is caused by the micro-organisms of the disease itself, or is caused by a subsequent infection from cocci which are normally in the respiratory tract. As Kanthack has put it—"The infection may be homologous or heterologous." P. Horton Smith of St. Bartholomew's Hospital summarizes thus—"All agree that the pneumococcus is by far the most frequent cause of primary broncho-pneumonia, and that next to this, though at a great distance, comes the streptococcus." In diphtheria, from the latest investigations, the broncho-pneumonia is simply a part of the disease, the lesions in the lung being caused by the Loeffler bacillus, often associated with the pus cocci which always accompany the infection in the throat. This is therefore an homologous infection.

The broncho-pneumonia occurring in influenza is now regarded in most cases to be a part of the disease, though at times it is aggravated by the presence of other organisms, chiefly the pneumococcus. W. H. Smith has recently reported an analysis of the autopsy records of the Massachusetts General Hospital of seventy-three cases of broncho-pneumonia, and it was found that five were due to the influenza bacillus. It was found once in a case of lobar pneumonia, but was considered a secondary infection.

He compared the clinical, pathological and bacteriological findings. All of these cases were patients past middle life. He found the type of the pneumonias to be usually lobular, frequently consisting of multiple foci, with the foci often far apart, with a tendency to involvement of the lower lobe of the left lung. This was found in five of the six cases. This last point has been noted by other observers. In no case was rusty sputum observed. In no case was the spleen found enlarged. The influenza bacillus was associated with the pneumococcus in all but one case. In one case the influenza bacillus and the pneumococcus were found in the sputum during life, and in one case only was leucocytosis mentioned, and then it was increased.

He states that clinically these influenza pneumonias may give few, if any, signs beyond a moderate degree of fever, and a few fine, moist rales more or less circumscribed.

The broncho-pneumonia following measles, pertussis and scarlet fever never can be well understood until more is known of the etiology of these diseases. The streptococcus, the aureus, or the pneumococcus, or combinations of these are the commonest. Numerous other bacteria have been found, but they are not constant factors in the lesions.

Broncho-pneumonia following typhoid is probably not due to the typhoid bacillus, but to an infection with the pneumococci, streptococci, etc., from the bronchi.

In the acute or sub-acute tuberculous broncho-pneumonia occurring in the course of tuberculosis the pneumococcus is almost always found associated with the tubercle bacillus, and these, Holt states, in children run a course of simple broncho-pneumonia. How much influence the pneumococcus exerts it is desirable to know on account of the serum treatment.

Osler mentions a pneumonic type of acute tuberculosis which resembles in its onset and physical signs a frank pneumonia. The sputum is relied upon to determine it.

From autopsy reports and clinical observations German writers have attempted to form some classifications according to bacterial etiology. Denny has reviewed some of these observations in the Boston Medical and Surgical Journal. Most American text-books, if they discuss it at all, say that there is not yet sufficient data. Finkler has written, more than anyone else, chiefly on the rôle of the streptococcus. He has described cases with the clinical picture of typhoid—septic cases—also an epidemic type which was very acute and fatal.

Wasseman, Weismayr, Finkler and Neusser have described cases which they consider streptococcus pneumonia —the streptococci being found in the sputum—with the

following symptoms: The onset often less abrupt, malaise feverish and chilly sensations, pain in the side and cough, purulent but not rusty sputum, septic facies. They run a long course, the temperature is irregular, and there is no crisis. The physical signs are late in appearing. The upper lobes are more often affected that the lower. The local process spreads about. Bronchial breathing, moist rales and dulness may persist for weeks.

The German observations in streptococcus pneumonias have not been confirmed at any length by English or American clinicians as far as I can ascertain. They are certainly suggestive. Denny states that the diagnosis of streptococcus pneumonia must be made from the sputum.

From fatal cases of pure pneumococcus infection we know that the clinical symptoms and signs vary within wide limits. At the present time we cannot, it seems to me from my own observations, make a diagnosis from the sputum. It is true that pneumococci, streptococci, staphylococci and Friedlander's bacilli are present in pneumonic sputum, but it is equally true that these same organisms are very often in the throat in health, and in the sputum of other diseases than pneumonia.

The literature on this subject is meagre in definite details, but it seems to me that in considering the sputum in pneumonia it is well to remember Netter's observations on the examinations of the saliva of healthy people. He found the staphylococcus almost constantly, the pneumococcus in 20 per cent., the streptococcus in 5.5 per cent., and Friedlander's bacillus in 4.5 per cent. He considered these observations as being under rather than above the mark.

I wish to include in this paper a report of two autopsies which I have recently made upon cases of pneumonia. The clinical and the bacteriological sides were of much interest.

Case 1. Male, twenty-four years old, entered the City Hospital in Dr. R. W. Greene's service. There was a history of a two weeks' illness before entrance. During this time he had a sore throat, signs of bronchitis, irregular chills, with considerable temperature, though he was not in bed but a small part of this time. On entrance, he had a reddened, swollen uvula, tonsils and palate, moist rales over both chests, but no evidence of consolidation. His sputum was thick, tenacious and blood-stained. Later his tongue became much swollen. A throat culture showed simply the pus cocci. After a few days of irregular, moderately high fever, with no chills, his temperature became sub-normal, by lysis, where it remained two days, followed by a high, irregular temperature, with marked evidence of septicemia. He died on the seventeenth day of his stay in the hospital, and about the thirtieth day of the disease.

At the autopsy the body was much emaciated. There was a most intense tracheitis and bronchitis, with a thick, yellowish muco-purulent exudate. Occupying nearly the whole of the left lung, especially the upper lobe, were large areas of consolidation, resembling lobar pneumonia, but in reality lobular in type. The other lung was crepitant and very extremely engorged, but microscopically showed no areas of broncho-pneumonia. There was a coexisting fibrinous pleurisy, especially marked over the consolidated area. There were numerous very small abscesses in the spleen and in both kidneys. The spleen, kidneys, liver and heart showed the usual signs of acute degeneration. There was no evidence of any chronic disease. Cultures from the pneumonic area in the lung, the heart's blood, the kidneys, liver and spleen, showed a most extensive growth of the staphylococcus aureus. This was the only organism present. Scraped slide specimens from the lung showed only cocci, not in chains. Recognizing that

the pneumococcus may be overlooked, a careful search was made for it, but none was found. It seemed to be an infection with the aureus alone. Whether pneumococci had been there in the beginning, I could not determine.

This case shows a very slow onset, with recurring chills, irregular temperature and sepsis, at the end suggesting pus formation, and illustrating the fact that a general aureus infection will, as a rule, if given time, produce abscesses.

Case 2. Female, fifteen years, well developed and nourished child; entered Memorial Hospital in the service of Dr. O. H. Everett. She had had asthma for eight years, and her mother had been an asthmatic for many years, and had died of pneumonia.

For two weeks before entrance she had a constant and severe headache, a slight cough and many asthmatic attacks. Four days before entrance the headaches grew worse; she was nauseated and took to her bed, but had no chill or chilly sensations. She complained of being feverish for several days. The sputum on the following day became streaked with blood, though for a day or two before it had been thick and yellowish; pain in the right side and coughing. On entrance her face was flushed, alæ nasi working, hurried respiration. There was dulness over the lower portion of the right lung, with bronchial breathing and crepitant rales. On the upper part of this lung the percussion note was somewhat tympanitic. Over the left lung there was no dulness, but the respiration was harsh and attended with a few moist rales. The white blood count was 9,200 per cmm., *i.e.* without leucocytosis, a fact which is often observed in fatal cases. I examined the sputum and found a few pneumococci and many streptococci. The urine was fairly acid, with a specific gravity of 1014; no albumen or sugar. The chlorides were somewhat diminished, but not absent. She was evidently very septic, and was delirious during her stay in the hospital.

Three days before she died a peculiar eruption appeared on the chin, and in about twenty-four hours spread over the entire face, extremities and body. It was a very malignant-appearing eruption, resembling a severe form of measles upon a scarlet-fever background.

The autopsy showed an acute lobar pneumonia in a stage of red hepatization of the lower part of the middle and entire lower lobe of the right lung, with a fibrinous exudate covering the adjacent pleura. There was a large hemorrhagic infarct in the lower lobe of the left lung. The spleen weighed 180 gm. All the organs showed the usual acute degenerative changes. Pneumococci and streptococci by culture and by smear preparations were found in large numbers in the pneumonic areas, and streptococci alone in the heart's blood, the spleen, kidneys and liver. Sections of the eruptions in the skin, hardened and stained, showed many streptococci in chains in the lymphatics, resembling erysipelas. Chains of streptococci were demonstrated in every organ in sections. Unfortunately the head could not be opened.

The onset of this case was gradual; there was no chill, the sputum was blood-stained, but not rusty. Delirium, sepsis, and a most peculiar skin eruption marked the course of the disease in the hospital. The physical signs and post mortem findings in the lung were those of an ordinary lobar pneumonia. Here we had a pneumococcus infection limited to the lung, and a general streptococcus septicemia. How much importance should be attached to finding streptococci in numbers in the sputum I am not prepared to state. The interesting phase of this case was the presence of the streptococci in the skin eruptions.

In conclusion, while we have much positive knowledge of the different single and mixed infections of pneumonia, we are not yet able to positively diagnose one infection from another by symptoms alone. The symptomology of

certain streptococcus and influenza cases seems to present a uniformity which is suggestive. The sputum offers an attractive field for laboratory research from a diagnostic standpoint and certainly gives promise of important results.

As yet the blood and urine give no reliable clue.

ARTICLE XX.

# LOCAL AND GENERAL TREATMENT IN UNCOMPLICATED SEVERE CASES OF PNEUMONIA.

## SPECIAL CONSIDERATION OF THE USE OF ALCOHOL AND OTHER STIMULANTS IN PNEUMONIA.

## DELIRIUM AND DELIRIUM TREMENS AS COMPLICATIONS OF PNEUMONIA.

By NORMAN F. CHANDLER, M.D.,
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READ JUNE 12, 1900.

# TREATMENT IN SEVERE UNCOMPLICATED CASES OF PNEUMONIA.

I HAVE no new or novel plan for the treatment of pneumonia. The question is an old one, and different plans and methods of treatment have been tried. These have varied as the idea regarding the true nature of the disease has changed. At present it is quite universally believed that the disease is one of the specific infectious fevers, that it is an acute general disease with a characteristic local lesion. It is a matter of common observation that the ratio of the local inflammation and the general condition is by no means a constant one. The most alarming symptoms may be found when only a small patch of lung is involved, and it is not uncommon, especially in children, to have a very typical picture of the disease for some days before any infiltration into the lung can be demonstrated. On the other hand one whole lung may be solid, or there may be a double pneumonia with comparatively slight constitutional disturbance.

The disease appearing in epidemics as it does, often affecting a number of people in the same household, is evidence of its infectious character; and the facts which have been demonstrated by experimental work make the conclusion believable that pneumonia is a specific general disease and not a local one. Other facts which are generally accepted and which must influence the plan of treatment adopted are these:—the disease is self-limited, is of

short duration, and in uncomplicated cases the danger is from heart failure, whether brought about by local or general causes.

When dealing with a specific disease it is rational to seek a specific remedy for that disease,—either a drug that will destroy the cause or neutralize its action, one that will produce a definite result, like that of quinine in malaria, or an antitoxin that will do for the disease in question what the Behring antitoxin has done for diphtheria.

The question of serumtherapy it is not my province to discuss, but I think it very doubtful if there is any remedial agent, a drug, that can be relied upon to produce a specific action in this disease.

Treating the disease with sedatives, aconite or veratrum viride is warmly advocated by some writers, and as strongly opposed by others.

Theoretically the practice is not a good or safe one, because of the action on the heart, whose strength should be carefully guarded from the first. At the very beginning of the disease it may be capable of giving some relief, but most men believe the patient is safer without sedatives that depress the heart.

Petresco and others advocate the use of large doses of digitalis. From seventy-five to one hundred and fifty grains of digitalis leaves must be used in twenty-four hours to get the beneficial result. This seems an enormous amount of digitalis, but he claims the benefit is seen in lowering of the temperature, slowing of the pulse, sometimes to thirty or forty, and in an early crisis with no toxic effects. He reports 1192 cases treated with mortality of less than three per cent. His cases were soldiers, and therefore the most dangerous extremes of life and those ill from chronic complaints were excluded.

In the hands of other observers the plan has made a good showing. One observer found such toxic effects,

nausea, vomiting, vertigo, irregular pulse, that he felt forced to give up its use.

A specific action has been claimed for pilocarpin, sodium salicylate, iodine and chloride of calcium, etc., but the results obtained from their use have not been such as to lead to their general employment. The wide variation in the type of the disease and the conditions attending it make it difficult to prove the superiority of one plan of treatment over another, but when there are so many varying opinions it is probable that no very definite results have been reached.

Treatment by the application of cold to the chest is not new, but seems to be coming into more general use in recent years. Those who advocate this method of treatment do not claim for it a specific action, but they do claim that it gives a lower mortality, and that the crisis comes earlier when their plan is properly carried out. They advise covering the affected area of the lung with ice bags which are covered with towels. Soon after the ice is applied the temperature is lowered, the pulse and respirations become slower, and pain is relieved. Some writers claim that this plan of treatment does more than relieve symptoms,—that it really has a decided influence on the inflamed lung, that it contracts the pulmonary capillaries, improves their tone and thus favors resolution.

Dr. Mays of Philadelphia, in the Medical News, October, 1894, says, "How it can dissolve an exudate, or an infiltration, is not so clear to me. That it accomplishes this I am firmly convinced. For example, there is a pneumonic area which is wholly devoid of vesicular sounds, and has a flat percussion note and bronchial breathing, indicating beyond doubt that the process has passed beyond the stage of engorgement and into that of exudation or infiltration; yet the application of ice to this spot will in a remarkably short time develop a new group of physical signs, such as crepi-

tation, reappearance of the vesicular murmur, diminution of flatness. This has been observed by myself over and over again."

Dr. Mays reports 195 cases with a mortality of about three and one-half per cent. The number of observations is too small for any definite conclusions to be reached.

There being no specific remedy for the disease, the expectant or symptomatic plan of treatment gives the most promise of success. If we accept the facts, that we cannot cure the disease, abort it, or cut it short, the only rational thing to do is to treat the patient and help him to cure himself. Many cases of moderate severity do not need any treatment beyond good nursing, care in regard to diet and rest, with perhaps a laxative. The more severe cases demand careful watching, every effort being made to save the patient's strength to fight out the disease. The symptoms must be met when they arise, and they should be treated by such measures as will accomplish the desired end without doing ultimate harm.

Certain measures are necessary in every case, and they are no small factor in bringing about the desired end. *Absolute rest* in bed is to be insisted upon; sitting up to have the bed made, a common practice when there is not a trained nurse in attendance, sitting up in bed to take nourishment or for physical examination, getting out of bed to evacuate the bladder or bowels—all call for extra labor on the heart, and should be avoided as far as possible. *The room* should be large, if such can be had, well ventilated at all events, and this can be done if one is not afraid of taking cold, of which there is very little danger. If a man has only one lung with which to supply himself with air he needs that article in as pure a state as possible. The temperature of the room is best kept at from 65° to 70°.

*The diet* is another important care. The struggle in pneumonia is not a long one, and I think we are in danger

of feeding too much rather than too little. Light easily digested food, milk, broths, eggs and beef juice fill the requirements well when given in small quantities at intervals of two or three hours. In all the acute infectious diseases I believe water should be regularly prescribed. Four to six ounces every two or three hours keeps or helps to keep the kidneys active and to carry off the tissue waste caused by toxins of the disease.

The first few hours of the first day of the disease when it is of a severe type is often a most trying time for the patient and not an easy one for the physician. The sudden onset, the chill, the pain, the rapid rise of temperature, the quickening pulse and difficulty in breathing, make a profound impression on the nervous system and there is an urgent call for relief. It is not an easy matter for the physician to decide what is the best course to follow at this time. It must be a question of judgment, for no fixed rules can be laid down to meet every case. If the case is a strong full-blooded young adult, with a full bounding pulse, Dr. Osler believes bleeding is capable of doing good even to the extent of saving life. The effect comes quickly and the patient is relieved at least for a time. If done at all it should be done early. The profession are not unanimous in favor of this procedure, some claiming it never should be done, that a patient can be bled into his own veins with equally good results by using veratrum or aconite. Still others believe the occasion very rarely comes when either bleeding or the cardiac sedatives are demanded.

I think it a common practice with many physicians, and I know it is with myself, to use some preparation of opium in this stage of the disease. It has the sanction of Dr. Loomis and Dr. Osler, and unless the disease occurs in one who already has a bronchial secretion I see no objection to its use. Dr. Loomis believes that if a patient is brought under the full influence of morphine early in the disease he

is placed not only in better condition to resist the shock but also to combat the pneumonic process.

When the lung has become infiltrated, opium should be used with caution, because paralysis of the bronchi which it induces favors the accumulation of secretion and may add seriously to the trouble in breathing.

The great question from the beginning to the end of pneumonia is to support the heart. The right heart has an additional burden to carry in pneumonia, the solid lung acting to obstruct the pulmonary circulation, but this is not the great factor in causing heart failure. The poisonous toxins of this disease, like those of typhoid and diphtheria, acting upon the nerve centres governing the heart, are the source of greatest danger. We have no specific remedy with which we can meet this specific source of danger, but in spite of that fact much can be done to lessen it. By beginning early in the disease, relieving pain, promoting rest, careful feeding and nursing, in fact doing everything possible to save strength, and nothing to weaken the vital forces, the patient is much more able to withstand the effect of the poison which is overcoming him; and although we cannot influence the production of poison or neutralize it, we can do much toward eliminating it. The skin and the kidneys are the organs to which we look for help in other toxic conditions, and they are capable of doing much for one with pneumonia. Water outside and in I believe to be the best therapeutic aid to an elimination of toxic products. The details for carrying out this valuable measure will be explained by Dr. Baruch, to whose work I am indebted for instruction in this matter.

Medicinal agents may not be required, but if the heart is found to be losing force we have valuable cardiac tonics which will help tide us over the critical time. Strychnia and alcohol I believe to be the most effective. In the use

of these stimulants we should not wait too long, for it is easier to keep up the action of the heart than it is to bring back its power when too far gone. I am in the habit of giving strychnia early in the course of the disease, gr. 1-50 every four hours, for its tonic effect, and this is increased to gr. 1-30 or 1-20 every three hours, often given *sub cute* if the case seems to demand it. All are not agreed on the question of the use of alcohol, but if it is ever a valuable therapeutic agent, and I believe it is, it is in just such toxic conditions as the acute infectious diseases produce. It should not be given as a routine practice, nor should it be used carelessly, but if needed it should be used in such amounts as the case demands. It should be given when the pulse becomes rapid, small, feeble and compressible, and the amount needed must be determined in each case by watching the effect; sometimes a few ounces of whiskey or brandy will be found sufficient, while in another case the amount may have to be increased to twenty or thirty ounces in twenty-four hours before the desired result is gained.

Digitalis is advised as a cardiac tonic, but is not so generally used as strychnia or alcohol, and my experience with it has not been satisfactory. When the right heart is distended, nitro-glycerine is valuable as a heart tonic, and in case of collapse ether *sub cute* may be used.

The pain, which is due to pleurisy, is a very troublesome symptom and one of the first we have to meet. It is increased by cough and deep breathing and the patient instinctively limits the expansion of the chest as much as possible. If the pain can be relieved the rapid breathing and dyspnœa are in a measure relieved. To accomplish this, local applications are necessary. Hot or cold applications will often give temporary relief. If the patient is strong enough to permit the use of leeches they may be tried with promise of success; but if these fail, morphine subcutaneously will usually give relief from pain and at the same time quiet the distressing cough.

Delirium in uncomplicated pneumonia is common, and is the result of fever, weakness or toxemia. Coming early in the disease in the young and vigorous, I have not found it of special importance, except that its sudden appearance may lead to a wrong diagnosis and the true nature of the disease be overlooked. I have never found it to require special therapeutic measures,—those which are indicated to relieve the general condition being sufficient. When due to weakness, as is often seen in the aged and dibilitated, some form of alcohol is strongly indicated. It is a most unfavorable sign.

Delirium tremens is a serious complication of pneumonia, indicating as it does a nervous system already weakened by a systemic poison. Delirium tremens alone, without pneumonia, often taxes the strength of a patient, and when it comes as a complication of that disease the result is, as one would expect, often fatal. The daily amount of alcohol should not be cut off, and it is often necessary to increase it. Sedatives are required to promote rest and sleep, and chloral is the most effective in alcoholic cases.

Persistent high temperature calls for active treatment, and I believe the safest and most effective measure with which to meet this condition is cold applications, either sponging with cool water, the ice bags, or cold compress to the chest as advised by Dr. Baruch. The medicinal antipyretics are too depressing and unreliable to be admitted in the treatment of a disease where so much depends upon the heart. Temperature below 105°, with a good pulse, is cause for less anxiety than a low temperature when the heart shows sign of weakness.

## DISCUSSION.

Dr. E. O. Otis, of Boston.—I think we are all agreed with Dr. Chandler that in dealing with pneumonia we are dealing with a toxemia, and a toxemia which manifests its malignant influence particularly upon the heart, causing cardiac asthenia and, as some German observers seem to have shown, also upon the vasomotor system, causing a paralysis of the vaso-motor nerves and, consequently, a dilatation of the arteries. One of the principal points in treatment is, as the doctor has indicated, restoring and maintaining the strength of the heart and tone of the vessels, and I think we are all agreed with him that the two principal drugs most available in doing this are strychnia and alcohol, the strychnia being used hypodermatically in not too small doses; and I would suggest as a valuable form of alcohol, Tokay wine. With regard to digitalis there seems to be a diversity of opinion among the profession and a still greater diversity with regard to nitro-glycerin. In a paper published by Elsner of Syracuse last year he made the statement that from a large experience he had never seen a case of pneumonia recover which had been vigorously treated with nitro-glycerin; and on the other hand, Dr. Walker of Philadelphia in discussing this paper said he had used nitro-glycerin with very satisfactory results, and cites one case of a child in which he gave one one-hundreth of a grain every two hours for a week with beneficial results. Dr. Andrew H. Smith of New York favors the use of nitro-glycerin under special circumstances, namely, when the right heart is embarrassed and engorged. He says the nitro-glycerin at that time still further paralyzes the vasomotors and dilates the vessels and relieves the heart in that way. Probably nitro-glycerin used in a routine way does more harm than good, but used to produce certain special effects it is of value. Then there are the diffusible stimulants which have always been used and I think always will be, and I believe are of material aid when frequently administered in cardiac asthenia. Two other drugs I would mention as of value, sulphate of spartein and caffein.

In regard to external applications I would add to Dr. Chandler's list that of dry cupping. The thing to be de-

voutly hoped for in the future in the treatment of pneumonia is an antitoxin which will combat the toxemia of this disease as the antitoxin of diphtheria does the toxemia of that disease, but until we do obtain this we shall have to continue to rely upon the expectant and somewhat empirical treatment of pneumonia, and that practitioner will obtain the best results always from his cases of pneumonia who most clearly at the outset comprehends the conditions of his especial individual case and its demands, foresees the emergencies which are likely to arise, and has thoroughly well in hand the knowledge of the drugs which he proposes to use and a clear perception of the effect which he desires to produce. Not long ago I was conversing with a physician from another city and expressed my desire to know how a distinguished practitioner had treated the case of a distinguished gentleman desperately ill with pneumonia. "I can tell you," he replied; "he simply gave everything that he thought would do any good," and practically that is about what we all do when we have to treat a desperate case of pneumonia.

Dr. J. F. A. Adams, of Pittsfield.—In the list of drugs that have been mentioned I notice one is omitted, and that is one in which I have begun to have a great deal of faith, namely, the salicylate of sodium. I have used it this past winter and suggested it to some of my friends, and I must say it promises very handsomely indeed. It is the only drug which in my experience has ever come near to acting magically in pneumonia. It seems to act very much as it does in rheumatism. You know what it does in acute rheumatism—stops the pain, produces diaphoresis, brings down the fever. It seems to do just the same thing in pneumonia. I have not used it long enough to speak with confidence about it, but I think it is worth trying. At the beginning of an attack put your patient upon full doses of salicylate of sodium; I give about sixty grains a day. Others have given as much as one hundred and twenty grains. Give strychnia with it, and I think you have a good combination. It reduces the temperature and does it safely. I believe there are great possibilities in the salicylate of sodium. I first got the idea from the article by Dr. Andrew H. Smith published in the Medical News for Dec.

16, 1899, in which he recommended this as well as the salicylate of ammonium and carbonate of creosote, and referred to the wonderful experience of Liegel with the salicylate of sodium among the miners of Leoben-Seegraben. I used it immediately in some extreme cases and got very remarkable results; that is, within twenty-four hours after beginning the doses the temperature came down, the inflammatory process was arrested, the crisis had come. I don't say it was a case of propter hoc, but it looked very much like it.

ARTICLE XXI.

# PROGNOSIS OF PNEUMONIA, AND TREATMENT OF THE FAMILY.

By JAMES W. HANNUM, M.D.,
OF LUDLOW.

READ JUNE 12, 1900.

## PROGNOSIS OF PNEUMONIA, AND TREATMENT OF THE FAMILY.

Our interest in the Prognosis of Pneumonia is accentuated and made serious by reason of the high mortality rate at certain periods of life and in particular types of the disease.

Age is the most potent factor in determining the issue of life or death in pneumonia. While children, as a rule, are conducted safely through an attack, the majority of patients who have passed their fiftieth year fail to recover.

Before the age of one and a half or two years, lobar pneumonia is believed to be somewhat rare, but in a number of well marked cases of primary croupous pneumonia under one year of age, observed by myself, the physical signs were unmistakable, and a considerable mortality prevailed. Some statisticians record one hundred per cent. of recoveries in all cases under five years of age, as well as in those from five to ten years of age. Except in a selected series, it is probably nearer the fact to estimate a mortality of from two to four per cent. in children under ten years of age. In the second decade of life there is a tendency to a more extensive pathological change, which increases the death rate to eight or ten per cent. Between the twentieth and thirtieth years nearly one of every four dies—twenty-five per cent. Of those from thirty to forty years of age from sixty to sixty-five per cent. recover. Between forty and fifty years, fifty-five to sixty per cent. recover. Pneumonia after sixty is the most fatal of all acute diseases, and

it has been estimated that ninety per cent. of all deaths after the age of seventy-five years are due to pneumonia. Johnson might well have had this disease in mind when he wrote:

> "Superfluous lags the veteran on the stage
> Till pitying Nature signs the last release
> And bids afflicted worth retire to peace."

The force of Walshe's observation relative to the striking difference in the number of deaths at different ages is readily comprehended. He says: "There are periods of life when it is next to impossible to save—there are periods of life at which it is not easy with common prudence to lose —a pneumonia patient."

The influence of sex is seen in the larger number of males affected, while women succumb in larger ratio.

Cases in autumn and winter show a slightly higher mortality rate than in spring and summer.

The alcohol habit has a marked effect on prognosis, a combination of delirium tremens with pneumonia affording little chance of recovery.

The extent of lung tissue involved influences the prognosis to a considerable degree. Some writers state that double pneumonia is rarely recovered from, others that nearly fifty per cent. get well.

Much depends upon the type of the attack. Unfortunately we have had opportunities in recent years of becoming familiar with pneumonia as a sequel to, or as complicating epidemic influenza, due, not to the diploccocus of fibrinous pneumonia, but to a mixed infection, constituting "adynamic," "migrating"—involving successively different portions of the lung, and "malignant" pneumonia—a type less amenable to treatment by drugs, and attended with a larger mortality, from thirty to forty-five per cent.

The character of the sputum in any case of pneumonia gives much information that can be utilized in determining

prognosis. The brighter the color, the better the chances of recovery. Dark or dirty yellow sputa afford indications of a fatal termination. Absence of expectoration, as well as difficult and scanty expectoration, portends ill to the patient.

The coexistence of other diseased conditions has a disastrous effect in many cases, the prognosis in diabetic patients being extremely bad, though death is not always preceded by delirium and coma as has been generally taught.

Rheumatism, chronic nephritis, and diseases causing cardio-vascular changes, incident to the later periods of life, when complicating pneumonia, help to swell the fearful mortality and compel us to give an unfavorable prognosis.

Pneumonia when occuring as a sequel to measles is not usually fatal.

A temperature over 105°, a high pulse rate, or frequent respiration with low temperature, make the outlook dubious.

The prognosis in cases treated in hospitals is generally much more unfavorable than in private practice, an estimate for the former being from twenty to forty per cent., contrasted with from five to twenty per cent. for the latter. Many moribund cases, as well as the large number of alcoholics entered at institutions, have a disturbing effect on the mortality rate. In military hospitals the prognosis is much more favorable, the number of fatal cases reaching only from four to seven in a hundred.

More important considerations in prognosis are the condition of the heart, as revealed by the degree of clearness and strength of the pulmonary second sound, and the presence or absence of leucocytosis. *" With increased tension in the pulmonary artery there comes a sharper recoil and greater intensity of the valve sound in the pulmonary area, provided the ventricular contractions are not impaired. In this intensity we have practically a measure of the degree

* Dr. Andrew H. Smith. Twentieth Century Practice.

of obstruction to the blood current in the lungs. It is often easy to observe, as consolidation progresses, the constantly increasing sharpness of the pulmonary second sound, and in favorable cases this accentuation, after reaching a certain point, is maintained with little change until resolution commences. After the circulation becomes easier the valve sound loses its intensity just in proportion as the pulmonary condition improves. Loss of accentuation does not always depend upon relief of the pulmonary circulation. It may signify a weakening of the right ventricle, and a lessened power to force the blood into the artery, which comes when the right heart begins to flag from excessive labor, or because of the depression arising from the specific infection. In either case when it is found that the pulmonary second sound is becoming weaker without any evidence of a favorable change in the condition of the lung, we may draw the conclusion that the right heart is failing, and that the tendency of the case is towards a fatal termination. The radial pulse gives only indirect and inconclusive evidence relative to failure of the right heart. The right ventricle may be on the verge of exhaustion, while the left retains almost undiminished vigor; the radial pulse will then simply indicate that less blood than usual is flowing beneath the finger. I have never seen disaster come in a case of pneumonia unless it was preceded by a period in which the pulmonary valve sound was progressively losing in intensity. I have never seen a case with sharp and distinct pulmonary valve sound in which a favorable prognosis as to the immediate future was not justified by the event."

"The weakness of the right heart, that is indicated when the valve sound becomes less intense, takes place" says Dr. Smith, "chiefly in two classes of cases. First, that in which the system is overwhelmed by the virulence of the infection. The cases are marked by a severe and protracted chill,

great muscular prostration, a small and relatively weak pulse, and marked involvement of the nervous system. We may well believe that in such cases the heart muscle is poisoned, and that consequently the right ventricle, with the increased labor thrown upon it, early becomes exhausted. Such cases are apt to prove fatal before defervescence takes place. The second class of cases is made up of those in which, from age, intemperance, previous illness or other cause, the integrity of the circulatory apparatus is compromised in advance. Here we have a constant tendency to the invasion of fresh areas of lung and to the supervention of œdema. This tendency, while it increases the labor required of the ventricle, is itself increased in proportion as the ventricle weakens under its burden."

Recent investigations upon the subject of Leucocytosis as it relates to the prognosis of pneumonia have developed much that may prove profitable, as well, through the employment of remedial measures, having as the object of their administration the production of an increased number of white blood corpuscles.

It is found that "high temperatures carry with them (in general terms) a large number of leucocytes. From a prognostic point of view this is of great importance, whether we consider the leucocytes as scavengers, and assume that they destroy the cocci, which are the medium of infection, or whether we assign to the white cells a share in the production of antitoxin, certain it is that severe cases in which there is marked leucocytosis do better as a rule than similar cases in which the increase of white cells is relatively slight. When the leucocytosis is inconsiderable it will generally be found that the system is not reacting energetically against the infection, a condition which suggests an unfavorable prognosis. On the other hand there may be a high degree of leucocytosis which is not due to the pneumonia pure and simple, but to some complication, as pleurisy or pericar-

ditis, and if this is not taken into account, a faulty prognosis may result. An examination of the deaths will show that in severe forms of lobar pneumonia a slight leucocytosis is a very unfavorable sign. In six of Emery's fatal cases, the number of leucocytes was subnormal. Not one case recovered in which the disease was of even moderate severity when the number of leucocytes fell below 14,000. In several instances again a slight leucocytosis seemed at the time the only unfavorable prognostic sign in cases ending fatally."*

It would seem, then, that we have in the microscopical appearances of the blood a reasonably certain indication of the gravity of the disease, and the chances of recovery.

Another observer says that "both clinical inquiry and laboratory experiments have shown, that leucocytosis is usually a favorable sign in pneumonia. This is well established by the studies of Limbeck, Billings and others, who find that an active, inflammatory leucocytosis during fever indicates a good reaction, and while not invariably so, is often a good prognostic sign. Absence of leucocytosis is in any case usually a bad omen.

Kanthack and Lloyd declare diminished leucocytosis with falling temperature to be a favorable sign, while a continuous high temperature, with a small number of leucocytes, is unfavorable. A persisting or increasing leucocytosis with fever is favorable. A satisfactory prognosis is not established by the presence of leucocytosis, but it is a most valuable indication when taken with associated temperature, the curves of which are often roughly parallel."†

While not strictly within the province of this paper, I ask you to note, that Naegli-Akerbloom gives‡ to large doses of the infusion of digitalis, when exhibited according to Petresco's method, the property of inducing a rapid

* A System of Medicine. Allbutt.
† Dr. Dyce Duckworth. Lancet, 1896.
‡ Hare's Therapeutics.

hyper-leucocytosis, achieving, in my opinion, a result in aborting pneumonia comparable only to the effects of antitoxin in diphtheria.

A word only upon the second part of my subject: Treatment of the Family. The only tenable and justifiable course in our relation with other members of the household is, to be honest, candid, sympathetic and confidential. Let the friends of the patient know, so soon as the prognosis is reasonably clear, just what the course of the disease is likely to be. In managing these matters much will depend upon the temperament of the individual physician, but we may well study to correct a propensity to give a more favorable prognosis than the conditions will warrant, as well as to sedulously avoid inflicting unnecessary pain by making an unqualified declaration that a given case is hopeless at so early a stage in the attack as to preclude an accurate forecast of events. It is inevitable that blame must sometimes rest where it is not deserved, yet I know of no disease or occasion where the opportunities for professional aggrandizement equal those that are offered to us in ministering to our pneumonia patients. It were futile to expect always to escape criticism and censure, in cases of severe illness, involving responsibilities to be met in no other walk of life, yet he who is actuated by the traditional impulses and motives of the medical profession cannot fail to merit and to receive a large measure of emolument and adulation.

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## DISCUSSION.

Dr. C. F. Withington, of Roxbury.—During the last term of my service at the hospital, being possessed of the opinion which was expressed in this paper and is generally held, that a leucocytosis was a fair basis for prognosis in any given case of pneumonia, I had a rather large number of

observations made from day to day in thirty or forty cases of pneumonia, and to my great surprise the results did not bear out the statement so generally made in regard to leucocytosis, in regard to prognosis. The amount of leucocytosis seemed to be rather proportional to the gravity of the infection and not necessarily to the way in which the organism was reacting to that infection; that is to say, there were numerous cases in which leucocytosis was very high, ran to 30,000 or more, in which the patient died and in which the evidence of great systemic infection was strong, but on the other hand there were a few cases in which pneumonia was evidently present, the physical signs being clear and in which the amount of leucocytosis was very small, in fact none at all in one or two cases, and some of those cases did not die. For instance, a hasty summary of results shows that of cases having a white count exceeding 30,000, three recovered and two died; of cases from 25,000 to 30,000 whites, three recovered and one died; of cases from 20,000 to 25,000 whites, four recovered and none died; of cases from 15,000 to 20,000, twelve recovered and four died; of cases from 10,000 to 15,000, eight recovered and three died. Of cases showing less than 10,000 whites I am unable to give a full comparative statement, as some of the counts were doubtless made late, *i.e.* perhaps after the crisis. I find two cases, however, in which the maximum count during the height of the disease was 7,000 and 7,600 respectively, both of which recovered. On the other hand the highest count of the series, 42,000, was in a fatal case.

These observations, limited as they are, seem to show that a high leucocytosis, like a high temperature, represents the reaction of a typical pneumonia, and is sometimes, but not necessarily favorable. While a low count, like a low temperature curve, may represent a condition of great prostration and gravity, or on the other hand one of relatively mild infection.

I want to say a word or two about abortive pneumonias, which have not been spoken of. I know of no way in which we can tell what cases are going to prove abortive, and I do not believe any treatment we may institute has anything to do with the abortion of the disease, but such cases do occur in which the fever starts in with every indication

of great severity, physical signs of beginning pneumonia, and in which on the third or fifth day the whole process comes to an end and premature defervescence takes place, the patient being restored to health. I had a personal experience of that two or three years ago in having a pneumonia start Sunday with chill, temperature 104°, bloody expectoration and pain with marked relief by Wednesday morning, the return of symptoms on the other side which I believe were coincident with physical signs on the other side occurring on Wednesday afternoon and a practical restoration to bien aise and convalescence on Friday. Such a course happens not very rarely, but what relation treatment may have to such a happy shortening of the usual period of pneumonia I do not know.

ARTICLE XXII.

# CONTAGIOUSNESS OF PNEUMONIA.

By JOHN N. COOLIDGE, M.D.,
OF BOSTON.

READ JUNE 12, 1900.

# THE CONTAGIOUSNESS OF PNEUMONIA.

We know that many agencies are concerned in the causes of pneumonia, and that they play a most important part. We also know that certain of these are associated with special types of the disease. But among them all is one which is essential, one without which we believe pneumonia to be impossible—namely, the presence and activity of bacteria. It is generally the pneumococcus, but may be one of several, and is sometimes the association of two or more kinds. Long before the discovery of these organisms men noted features which led them to class pneumonia among the acute infectious diseases; and while considering the importance of atmospheric and other influences as causes, were not satisfied that these were all. What they observed and recorded is of the greatest value, but recent work has added to it and given some explanations.

The question of contagion in this disease has arisen many times, even though not commonly suggested. But pneumonia has occurred as almost a scourge, and this occasional epidemic form has received much attention. Instances of it are recorded from America, Europe and Asia; at all latitudes, from India to Iceland. Prisons and jails and barracks, city streets, tenement houses, and other over-crowded places, have suffered most; but villages, ordinarily healthful, and houses well situated and well cared for, have sometimes been visited.

[1] In the spring of 1874, at the Kentucky State Prison at Frankfort, seventy-five cases occurred among 735 inmates.

Pneumonia had been unusually prevalent in the section, due, it was thought, "to the excessively cold winter or some mysterious epidemic influence." But this had ceased; cases were rare; in fact, Frankfort and vicinity were unusually healthy at the time. For more than one-tenth of the population to be attacked in this short period suggests contagion, but is by no means proof, as will be seen later. Twice before has this prison had a similar experience. Epidemics have twice occurred in the prison at Christiana, Sweden. In 1880 at d'Ansberg, there were one hundred and sixty-one cases, with forty-six deaths in a period of five months.

[2] During the civil war, in 1864, pneumonia broke out among the six regiments of colored recruits at Benton Barracks, Missouri. It raged from about Christmas time until the last of April, when it suddenly stopped. From January 1st until May 1st there were seven hundred and eighty-four cases treated in the hospital. One hundred and fifty-six of them died. Men occupying the same bunks with those affected were very much more likely to be attacked than those remote. Physicians and nurses suffered from it. Dr. Ira Russell, whose description of this epidemic is contained in the Memoirs of the United States Sanitary Commission, writes that: "Many of the most intelligent surgeons were led to believe that the disease was actually contagious." [3] Ziemssen says that among soldiers pneumonia is always more prevalent in garrison than in field duty.

[4] An epidemic on some ships of the Mediterranean fleet was described by Dr. Brayton, R. N., in 1864. He states that there were good grounds for believing that the affection was communicated by the sick landed from the vessel to other patients in the Malta hospital.

What is true of other infectious diseases is also to be true of pneumonia, *i. e.* that over-crowding favors its

spread. It also favors its severity. [3] In Paris the number of deaths among the nuns from this cause was nearly six times as great as among the washerwomen.

[5] As a most interesting illustration of village epidemics may be mentioned one occurring at Riethnordhausen situated on a slope in hilly country, higher than any surrounding villages. The number of inhabitants was seven hundred. Forty-two cases of pneumonia occurred, twelve among the children in the school house where it started. In the families or among the intimate associates of these twelve children, were twenty-six more. The remaining four were only indirectly exposed by three individuals healthy themselves, but who had been in contact with the sick.

House epidemics are not rare and doubtless instances will occur to many of you. [6] Osler tells of seeing three in one family consecutively attacked by the most malignant type of the disease. He says that as many as ten residents in one house have suffered. In hospitals those in beds adjacent to pneumonia patients are said to have acquired the disease.

In nearly all the illustrations cited it is plain that other agencies than bacteria have played a most important part. The village epidemic is the clearest exception. Such factors, however, may all be classed under one heading called de-vitalizing influences; sometimes one thing, oftener many. In the case of Frankfort prison, there were seven hundred and thirty-five men in six hundred and forty-eight cells. The ventilation was absolutely bad, and the filthy habits of the prisoners only added to the result. Five-sixths of the cases were from the upper tiers of cells. The winter was excessively cold, but this was probably of less importance. At Benton Barracks it was not a very different story. One hundred men were crowded into rooms meant for fifty. Here, however, an epidemic of measles was a most serious complication. In the

house epidemics, often much the same conditions prevail; not always, however. When several in one family have suffered, exhaustion from care and self-sacrificing devotion must be borne in mind. It is these house epidemics which are most suggestive of contagion.

Thus we have the way prepared and the soil made suitable for the bacteria. But before contagion in importance comes another means of infection, namely *auto*-infection. [7] This will probably account for most cases and plays a very important rôle even in epidemics, for the pneumococcus is present in the mouths of many healthy people—according to Sternberg twenty per cent.—and in the sputum of nearly all chronic pulmonary affections. In most cases of croupous pneumonia this is the organism found, and the disease as seen in epidemics, though often having features of its own, is generally of this type. On account of the great probability of persons being infected from their own mouths, it must be seen that no illustration gives positive proof of contagion, strongly as it may suggest it, unless it is known that there was no opportunity for auto-infection. Such could very rarely, if ever, be determined. For further light, then, we must look to the study of the bacteria themselves. We must see if they live and retain their virulence outside the body. In the laboratory, on artificial culture media, they are most delicate organisms and must be transferred every day or two; even then they will sometimes die, and will often lose their pathogenic properties. But outside the body, in deposits of sputum, they may live and remain virulent for some time. [8] They have retained this property in a sealed glass tube for over a year. [9] Silk threads impregnated with blood containing pneumococci, fragments of spleen and pneumonic sputum have been found virulent, as shown by experiments on animals, after thirty-two days of drying. [6] There seems to be a difference in resistance depending upon the source;

sometimes drying will destroy in two days, sometimes not in two or three months. [10] Welch says "Pneumococci in blood and sputum are in general tolerably resistant to drying, living and retaining their virulence sometimes for four months, though they may die much sooner." [11] Pneumonic sputum on linen cloths dried in the air and exposed to diffused daylight has kept its pathogenic properties for periods, varying with the specimen, from nineteen to fifty-five days.

Thus it is seen that the pneumococcus, the pathogenic agent of most pneumonias, will live for a considerable time outside the body in sputum and blood. It long outlives the cultures, due, probably, to its albuminous protection. [11] Direct sunlight, however, will usually destroy these bacteria in a short time, though twelve hours has not always been sufficient. A healthy person's saliva, containing pneumococci, is less a source of danger than pneumonic sputum, because it affords less protective covering against sunlight and drying. As regards insects as carriers, very little, if anything, is known.

Relying upon these observations, it seems safe to say that external infection is possible, and that pneumonia may be transmitted from one person to another by means of the sputum. It is contagious in the same way as pulmonary tuberculosis, though in a far less degree. Thirty observations are mentioned by Netter [12] in which well persons were attacked with pneumonia from having slept in the same bed with the sick. Bed-clothing and furniture seem occasionally to have been the carriers. As regards a third person as the means, nothing very definite can be said, though some strongly suggestive cases are given by Netter. [12] Neither can it be positively stated when, in the course of the disease, this danger is passed. [10] Usually after the crisis there is death of many of the bacteria and a loss of virulence, but this is not constant and the bacteria may be present and virulent long after recovery. [13] An-

other occasional source of infection is in the milk of a mother with pneumonia. This is confirmed both by clinical and experimental evidence. Intrauterine infection has occurred, but it is probably rare.

Of the other bacteria occasionally occurring in pneumonia, the bacillus of Friedlander is important. This is very resistant and will live for many months outside the body. There is evidence that in the epidemic at d'Auzberg it played an important part. Something at least very closely resembling it was found in the dust from the floor.[12]

The following are the conclusions drawn:

1. In the causation of pneumonia de-vitalizing influences are of great importance.

2. Most cases owe their origin to auto-infection, the bacteria being sometimes transient and sometimes almost permanent guests in the mouth.

3. The great mass of clinical observations, especially of village and house epidemics, point to contagion.

4. Experimental evidence shows pneumonic sputum to be a source of contagion.

5. Though contagion may be rare, and is usually very slight, it should be borne in mind by all intimately associated with the sick, and pneumonic sputum should be thoroughly disinfected.

[1] Am. Jour. Med. Sciences. Vol. 71, p. 76.
[2] U. S. Sanitary Commission Memoirs.
[3] Ziemssens Cyclop. of the Pract. of Med.
[4] Med. Times and Gaz. Jan. 23, 1864.
[5] Berliner Clin. Wochenschrift, Oct. 3 and 10, 1880.
[6] Osler's Practice of Medicine.
[7] Sternberg's Manual of Bacteriology.
[8] Kruse and Pansini.
[9] Netter. 1888.
[10] Johns Hopkins Bulletin, Dec. 1892.
[11] Bordoni-Uffreduzzi, 1887.
[12] Archives de. Gen. Med., May and June, 1888.
[13] Foa and Bordoni.

## DISCUSSION.

Dr. D. D. Brough, of Boston:—As to the question of pneumonia as an infectious disease, it is fairly certain now, and I think most of the later authorities take the ground that pneumonia is an acute infectious disease and that the statement of Fraenkel—that all cases of acute lobar pneumonia are due to the pneumococcus—has been established by recent investigators. I believe all the recent writers take that ground. With reference to the Board of Health the question comes up: Can we do anything with a disease which carries off so many people in the course of a year? In this city about 1,000 to 1,100 deaths occur every year from acute lobar pneumonia. Owing to the fact that we find the pneumococcus in all of the cases, the question at once arises whether a case of pneumonia should not be treated as diphtheria or phthisis is now treated; whether the Board of Health should not be notified, the patient be isolated, the house disinfected? But while theoretically and bacteriologically pneumonia is an acute infectious disease, yet I think practically it has not been proved to be so. Of course there is proof that many epidemics arise, and the arguments in favor of it being infectious are fairly conclusive; yet at the same time in this city for a good many years we have not had any epidemic of pneumonia where it was recorded as simply due from infection of one individual to another. I can find no authors who believe that the disease is directly communicated, and I think Dr. Coolidge has put it very fairly when he says it is on account of a devitalizing condition on the part of the patient rather than on account of direct infection. The fact that the disease is not communicable and that cases rarely if ever arise from patient to patient in hospitals, or from patient to nurse in outside practice, would make it inadvisable or inexpedient for the Board of Health to be informed and the person to be isolated. The fact that there is yet no definite proof that rooms are infected with the pneumococcus, notwithstanding that the pneumococcus will live in dried sputum for a long time, would make it inadvisable to use methods of disinfection afterwards. I think that the only ground we can take now is that while theoretically and bacteriologically we regard pneumonia as an infectious disease, yet from the point of view of the Board of Health it is not so in the sense we regard small-pox, measles and scarlet fever infectious.

ARTICLE XXIII.

# DELAYED AND PROLONGED RESOLUTION IN PNEUMONIA.

## SPECIAL TREATMENT. PROGNOSIS.

By STEPHEN A. MAHONEY, M.D.,
OF HOLYOKE.

READ JUNE 12, 1900.

## DELAYED AND PROLONGED RESOLUTION IN PNEUMONIA.

### SPECIAL TREATMENT. PROGNOSIS.

OSLER considers a delayed and prolonged resolution as a condition in which the pneumonic consolidation persists for four, eight or ten weeks with a gradual subsidence of the fever and a great improvement in the patient's general condition.

The causes which are usually assigned as being active in prolonging a pneumonic process beyond its usual duration are poor hygienic surroundings, run-down condition of the system, due to overwork, vicious indulgences or intercurrent disease, old age, inherited or acquired constitutional diathesis, especially the tubercular.

Since the discovery of pneumococcus lanceolatus and its etiological relation to this disease, many observers, especially the Germans, have attempted to explain all the clinical varieties of pneumonia, such as the bilious, grippe, malarial, typhoid, rheumatic and traumatic, as being due to the varied activity of the pneumococcus acting with one of the above mentioned causes or due to some other germ acting in conjunction with the pneumococcus. The other germs which have been found associated with the pneumococcus are Pfeiffer's influenza bacillus, Klebs loffler and comma bacillus and the staphylococcus aureus and the streptococcus pyogenes. Of these various organisms the streptococcus, according to many observers, plays a most important part in modifying the ordinary course of a pneumonia. Sometimes even it has been the only organism found.

In a letter, Dr. Andrew H. Smith of New York writes "that for some time past he has been teaching that defervescence by crisis indicates infection in which the pneumococcus lanceolatus predominates and that resolution by lysis occurs where the streptococcus or other organisms predominate."

In the German schools, for many years, they have differentiated their pneumonias according to their bacteriological findings. The pneumonia is a diplococcus or streptococcus according as the diplococcus or streptococcus predominates. In all cases in which the resolution has been delayed or prolonged the streptococcus has been found either alone or in conjunction with the pneumococcus. Many series of such cases have been reported by Leyden, Fraenkel, Weichselbaum, Neusser, Finkler, Harbitz, Weismayr and many others. Consequently the bacteriological examination of the sputum is of great diagnostic and prognostic importance in every case of pneumonia.

In looking back over my cases of pneumonia I find but three that may be properly classed as having a delayed and prolonged resolution. In these the initial symptoms differed but little from that of an ordinary frank pneumonia. The fever, chill, cough and pain in the side were present, perhaps not quite as marked as usual. The sputum, while blood streaked, never became "prune juice." It was not the thick and viscid sputum, clinging to the side of the vessel, but more purulent. The run of the fever differed but little from usual except in the later stages, when it became irregular and more like a septic fever. The physical signs in the lungs were late in appearing, consolidation not being complete until the second or third day. In all three of my cases consolidation was at the apex, two involving the left and one the right lung. The greater part of the remainder of the lung became involved before complete resolution occurred, but in my cases the part of the lung which was

first involved cleared last. The most characteristic feature, however, was the long duration of the physical signs.

In my cases, No. 1, Miss F., school teacher, twenty-five years old, still had dulness, bronchial breathing and moist râles at the end of the fifth week. The evening temperature did not disappear until the forty-fifth day.

In case No. 2, Mrs. M., forty-five, housekeeper, the physical signs of pneumonia persisted for nearly fifteen weeks. In this case, however, a lung abscess was detected in the eleventh week, which was opened and drained in the axillary space between the fifth and sixth ribs. She made a good convalescence.

Case No. 3, Mr. M., single, age thirty-eight, merchant, became convalescent from a case of pneumonia in the eighth week. The physical signs, however, persisted long after the temperature was normal. A winter spent in the South improved his general health, but still dulness and bronchial breathing with a few fine, moist râles persisted six months after the attack. A second winter spent in the South, however, cleared up the diseased apex, and the gentleman is in active business to-day.

It is seen, even in this small series of cases, that the duration and symptoms differed widely in each case. Unfortunately in only one case was a microscopic examination of the sputum made for germs other than the tubercle bacillus, and in that both pneumococcus lanceolatus and streptococcus pyogenes were found. While the cases differ widely clinically, I have no doubt that the multiform symptoms were due to an invasion by the streptococcus either at the beginning or during the course of the disease.

In regard to treatment: When a pneumonia has failed to resolve by the end of the eleventh or thirteenth day new conditions enter into the case which must be met by certain changes in our treatment. While heretofore all our treatment has been directed to sustaining the vital energies until

the expected crisis should take place, on some day between the fifth and the thirteenth, we now have to deal with a disease of an unknown duration, with a debilitating fever and a liability to various complications.

In order to fulfil the various indications for treatment the patient must be subject to the best hygienic conditions. Special care must be taken during the disease and convalescence that he should not be exposed to the tubercle bacillus. While pulmonary tuberculosis and a delayed and prolonged resolution are not etiologically connected, there is no doubt in my mind that a streptococcus pneumonia leaves within the lungs a culture medium in which the tubercle bacillus will very readily grow. That the streptococcus and tubercle bacillus may grow simultaneously in the same lung is an every day occurrence in consumption.

The hygiene of the body is of great importance. Rubbing and cool and tepid bathing at stated intervals not only reduce the fever but impart to the patient a sense of comfort which does much towards quieting and strengthening the nervous system already shattered by the hard initial stage of the disease.

A very important part of the treatment is the dietetic. Anything which interferes with the proper absorption of nourishment into the system directly threatens the patient's life. Yet great care must be exercised that you do not force nourishment to such an extent that you will hamper the digestive system in the proper performance of its functions. Sometimes the artificial preparations of beef and milk from some one of the reliable pharmaceutical companies will aid in maintaining a proper degree of nourishment.

Alcoholic stimulants should be exhibited according to indications arising from the condition of the heart and digestive apparatus. Over-stimulation is to be carefully avoided because of its deleterious effect on digestion.

Knowing that a pneumonia with a delayed resolution is due to the streptococcus pyogenes and that the centre of in-

fection is a local consolidated area in the lung from which the entire body is infected, the rational treatment seems to be one which will be directed towards destroying or inhibiting the growth of the germ in the infecting area. While there are many substances which will destroy the germ outside the body, there is as yet no known substance which, harmless to the living tissues, will accomplish that end with any degree of certainty in the living body.

Streptococcus serum was tried in No. 2 of my cases, but with absolutely no benefit.

In the writer's hands, pure beechwood creosote has given the best results. While in antiseptic power nearly equal to carbolic acid, yet its dose can be successively increased until even five hundred drops are taken within twenty-four hours. Moreover, the vapor can be inhaled, either from a sponge or mixed with steam from one of the various atomizers on the market.

The dose of creosote which an individual case will take varies greatly. In some cases gastric disturbances will be excited by doses of five to ten drops; while many will take with no apparent discomfort doses of from thirty to forty drops three or four times a day. In order to be effective, in my opinion, it must be given in as large doses as the stomach can stand, and must be continued during the entire course of the disease. It is my custom always to begin with small doses and gradually increase until the tolerance of the patient's stomach is reached. I usually give it in milk or capsule.

In conjunction with the creosote treatment minute doses of arsenic, either in the form of liq. potas. arsenitis or arsenious acid, is an exceedingly valuable adjuvant.

In some cases the iodide of potass, gr. v. to x., and corrosive sublimate gr. 1/40, apparently hasten the absorption of the exudate. However, the latter have not given as much satisfaction as the arsenic.

No matter what the drug treatment may be, one will occasionally encounter a case in which complete resolution will not take place. A slight dulness with bronchial breathing and fine moist râles still persist. The patient has a hacking cough with slight or no expectoration except in the morning, when he has a moderate amount of muco-purulent expectoration. No tubercle bacilli can be found in the sputum; the fever is irregular, usually very slight, but the patient does not improve. It is here that a change of climate will finally complete the cure. A residence in a high dry climate, or winters spent in the South, where the patient can be constantly out of doors, will cause a lung consolidation due to delayed resolution from pneumonia to disappear.

### PROGNOSIS.

The prognosis is much more favorable than in the original disease. In a series of 418 cases of pneumonia collected from various sources, in which a delayed resolution occurred fifteen times, all recovered but one. In the fatal case diabetes complicated it. Death occurred on the nineteenth day. This, however, must not be taken to mean that all cases of streptococcus pneumonia recover. Far from it. These statistics are taken from clinically observed cases of delayed and prolonged resolution. The initial stage had already passed. Moreover, many cases come to autopsy in the lung of which the streptococcus is found; also many German observers have reported epidemics of pneumonia, which pursued a very active and a very fatal course, in which the streptococcus was discovered.

### RESUME.

When a prolonged and delayed resolution occurs in pneumonia, it is due to a mixed infection usually with streptococcus pyogenes.

There is no specific treatment for this particular variety of pneumonia. A drug must be used, which will inhibit

the growth of the organisms in the lung, and in my hands creosote in conjunction with vigorous supporting treatment has given the best results.

The prognosis is much better than in the ordinary form of pneumonia.

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## DISCUSSION.

Dr. Henry Jackson, of Boston:—In looking over such cases of delayed resolution as I have had in private and hospital practice I have found the course of the primary disease to be very similar to the cases which Dr. Mahoney has described, namely, a rather atypical course from the beginning. I think I can add nothing to what Dr. Mahoney has said of the treatment and the general course of cases of unresolved pneumonia. To me the most interesting feature about these cases is the diagnosis. Dr. Mahoney has mentioned the question of tuberculosis which always arises in a pneumonia where resolution does not take place. In hospital practice I see many cases simulating unresolved pneumonia which often for a considerable time are thought to be simply cases of unresolved pneumonia that eventually turn out to be, and probably are from the beginning, empyema. In empyema in which there is a very large amount of pus filling the chest the diagnosis is easy, but cases with a small amount of pus in the lower part of the chest may well be mistaken for unresolved pneumonia unless we have resort to the needle. I have had three such cases in the last spring. The main points which induce me to aspirate are a diminution of the vocal fremitus and vocal resonance at the lower part of the chest. In such cases even if I hear respiration, a faint respiratory murmur transmitted almost to the base, if I find a diminished fremitus and diminished resonance, I think it far safer for the patient to aspirate and make sure. In aspirating it is necessary to use a pretty large needle, as the pus in these cases is thick, often mixed with fibrin and somewhat gelatinized tissue so that it will not run through a small needle. If empyema is found, incision I consider to be the best treatment.

DR. F. C. SHATTUCK, of Boston :—I should like to ask the question whether fever is a necessary accompaniment of these unresolved pneumonias. I recall a case many years ago in a mulatto in which absolute consolidation remained for months in a lower lobe without fever. It finally cleared. The inference from Dr. Mahoney's and Dr. Jackson's remarks is that there is always a second infection in these cases, that they are not cases of pure croupous pneumonia.

DR. MAHONEY :—In one of my cases fever was entirely absent and the consolidation persisted nearly twelve months.

DR. SHATTUCK :—That answers the question and brings out the point.

ARTICLE XXIV.

# PECULIARITIES OF PNEUMONIA IN CHILDREN AND ELDERLY PEOPLE.

By ALFREDA B. WITHINGTON, M.D.,
OF PITTSFIELD.

READ JUNE 12, 1900.

## PECULIARITIES OF PNEUMONIA IN CHILDREN AND ELDERLY PEOPLE.

PNEUMONIA in children may be illustrated by the following cases:

A child fourteen months old has inflammation of the large bronchi—cough, moderate fever, but without rapid respiration or nervous symptoms. On the third day, respirations which have been 25 to 30 suddenly run up to 40 or 60; the temperature which has been 101 runs up to 103 or even 104; the face becomes anxious and pale; the alæ nasi move rapidly. What has happened?

Simply this: an inflammatory process has extended by continuity from the larger bronchi to the small, thence to the alveoli, terminating and surrounding the latter. A pneumonia has succeeded to a bronchial catarrh—correlatively the physical signs change. Until this moment the percussion resonance has remained sonorous, auscultation has discovered sibilant whistling rales mingled with finer or coarser subcrepitant mucous rales; but now evidences that the pulmonary parenchyma is invaded appear, for in addition to the mucous rales, careful examination finds over both lungs disseminated areas of dulness only to be detected by light percussion, and over these areas are heard whiffs of tubular breathing. The diagnosis is complete. As the case progressed, the child became very pale and dripping with perspiration, the temperature showed a remittent range varying from 99 to 100 in the morning, to 102 or 104 at night, the respirations rising from 60 to 80 and even to 100

at times. Throughout the course of the disease the cough was loose, and on the seventh day diarrhœa set in, apparently caused by the abundance of mucus swallowed, which a young child is unable to expectorate. There was nothing eventful in the remaining course of the disease; the symptoms gradually declined and convalescence was established at the end of the third week.

The treatment consisted of brandy and strychnin and the use of [1] carbonated siphon water to dissolve the mucus in the bronchial tubes whose abundance was the most salient feature in the case.

In contrast with this case, another having a similar onset developed signs of dyspnœa in the second week, the cough subsided, the child became more and more cyanosed and very restless. There was marked retraction of lower chest wall, indicating atelectasis. Liquid rales were heard over both lungs as well as disseminated areas of dulness and tubular breathing; obstinate vomiting set in, apparently induced by large doses of brandy, and the child died twelve hours after I was called. She had been under homœopathic treatment, which evidently made no such adequate provision for stimulus of the respiratory centre as the strychnin was fortunate enough to do in the first case.

A child may be already ill with some other disease when the respiratory system is attacked. Not only the pneumonia but the bronchitis preceding it is liable to be engrafted upon a previously existing disease. In measles a bronchial catarrh is a constituent part of the disease—one of the expressions of tegumentary irritation. In pertussis the bronchial catarrh is almost equally imminent.

In any infectious disease invasion of the lungs is possible; the diminished resistance to infections of all of the tissues facilitates infection of the bronchial mucosa and the extension of this to the lung. [2] Most broncho-pneumonias are due to mixed infections, and vital resistance to these seems to be more difficult.

In this form of pneumonia which is extremely frequent and much to be dreaded in young children, whose nervous system and heart are peculiarly susceptible to deoxygenation of the blood, patches of [3] atelectasis are constantly forming through plugging of the bronchi by mucus. Interference with aeration tends to lower temperature without improving the condition of the patient. Hence a fall of temperature, while respiration remains greatly accelerated, say to 60 or 70, is a very ominous sign.

[4] The predominence in children of the lesions of lobular pneumonia over those of lobar type has been attributed by West to the conditions which render the expansion of the lung irregular, and thence to irregular and diffused aspiration of the agent of infection. [5] These conditions, which depend upon the fact that in the child the respiratory function is acquired relatively late, and remains for a long time relatively incomplete, are: the yielding character of the chest walls, the weakness of the respiratory muscles, and the diaphragmatic type of breathing. In addition must be considered the relatively embryonic state of the large epithelial cells of the small bronchi and alveoli—easily prone to excessive proliferation—thus to catarrh—yet with these lobular peculiarities of lesion, when the infection is due to the pneumococcus, the clinical course may be undistinguishable from that of infantile lobar pneumonia, *i. e.* the onset is abrupt, often with a convulsion, the duration short, the defervescence sudden.

[6] The clinical picture of pneumonia varies at many angles of the disease. There may have been no preceding bronchitis, even for a few hours. Thus in the case of D. B., a child of five, [7] the prodromata had consisted in an acute follicular tonsillitis, the poison of the disease had very possibly entered through the tonsils, thence passing to the lungs. The illness began with chilliness, headache, and a temperature of 101, the respirations were only 25 and no

morbid sounds could be heard in the lungs. By noon the next day, the temperature had risen to 105, the respirations were 60, with an expiratory moan, the alæ nasi flapping—sure indications of serious morbid process in the lungs—and the child was semidelirious. In the evening the aspect of the case was that of meningitis; delirium marked, teeth tightly clenched, temperature 105; *but* in distinction from a meningitis, the respiration and pulse were both rapid, and on this account I inferred that the cerebral symptoms, though doubtless indicating severe meningeal irritation, were secondary to a central pneumonia whose physical signs had not yet appeared. During the night the child became comatose, and the temperature rose to 106, while at the same time the respirations were 78 and the pulse 150. Early in the morning the temperatnre was 105, the respirations 70, the pulse 140. There was still nothing to be heard in the lungs. To the terror of the already distracted mother, I ordered a bath, and with a good deal of difficulty suceeeded in overcoming maternal opposition, and placed the child in the water for ten minutes. The bath was not cold, being at a temperature of 90°, but thus still 15° below the temperature of the patient. While in the bath, the child opened his eyes and recognized his mother. Half an hour later the temperature had fallen to 103. Examination of the chest revealed tympanitic pulmonary resonance and a few crepitant rales in the upper right lobe. At 5 P. M. the temperature had again risen to 105 and the child was semi-comatose, but less profoundly so than in the morning. A second bath was given, with resulting decline of only one degree in temperature, but the mental condition was greatly improved. The following morning, tubular breathing appeared. Correlatively the central process seemed to be relieved, the temperature falling to 103, rising again to 104 that same evening, and dropping to 98.5 the next morning, the fifth day of the disease. It rose

again slightly before the final fall, the respirations becoming normal twenty-four hours later. [8-9] Schlesinger calls this form of defervescence characteristic of lobar pneumonia in children.

The absence of perceptible physical signs in the lungs during the first three days of the illness was a noticeable feature in this case. It is frequently observed and frequently misleads.

"A hurried shallow respiration," observes Delafield, "sufficiently proves that the capillaries of large areas of pulmonary alveoli have become engorged, encroaching on the air space, hence visible respiratory distress which cannot be overlooked, though the percussion resonance remain clear." The morbid process has begun directly in the parenchyma as can only be the case under the influence of a poison circulating in the blood or aspired in respiration. I ask myself whether a poison carried to the lung through the blood vessels in its root would be more likely to produce a central pneumonia than when it reaches the alveoli diffusedly by inhalation.

When the physical signs could be distinguished in the lungs of this child, the violence of the disease was not increasing but beginning to abate. The dilatation of blood vessels toward the periphery of the lungs associated with the spread of the inflammation should and did tend to relieve the intensity of the central congestion as an artificial counter-irritation would do.

[10]The meningeal form of pneumonia is much more common in children than in adults. Pneumonia of the apex, especially the right, is most often complicated with cerebral symptoms. Why this should be is a problem that has often been discussed. But there is really no need to look for a casual relation between the lesions of the lungs and the meninges. They are the coincident expressions of a general infection. The facility of high temperature in children may

possibly be one of the compensatory mechanisms of the organism for more rapidly destroying toxins which the nervous system is peculiarly unable to resist. For combating the danger of toxemia the integrity of the kidneys in children is much to their advantage.

The noteworthy peculiarities in an influenza pneumonia in a child of three were the early marked nervous symptoms, the picking at bed clothes, the sunken eye balls, and very rapid emaciation, during the short course of five days ending in recovery.

The pain in pneumonia in children often misleads by being referred to the abdomen, as in case of a boy sent to the hospital as a case of appendicitis.

The important thing about a pneumonia in children is not to classify it, but to know when it begins, to know that a convulsion or vomiting in children may be the equivalent of a chill in the adult; to know that the occurrence in a child of hurried and superficial breathing suffices to indicate the beginning of pulmonary inflammation. The danger of this symptom is not in proportion to the directness of the physical signs, but to the gravity of the rational, functional and constitutional symptoms.

The physical signs are slight while or when the pneumonia is central; or,

The toxemia may be serious though the local inflammation is limited; or,

The bronchial catarrh, obstruction of the air passages and atelectasis may suffice to kill by suffocation though the areas of true consolidation be moderate in extent.

Finally, physical signs may easily be pronounced slight if they are not carefully looked for, the physician being discouraged by the cries and resistance of the child.

[11]The observation is of old date that pneumonia in elderly people resembles the pneumonia of infancy both in symptomatology and in anatomical lesion.

The same fundamental condition of inequality of the expansion of the lung is brought about in old people by the rigidity of the chest wall, the weakness of the respiratory muscles, and the tendency to catarrh by lack of vitality in the epithelium.

There are three main forms:

[12]First. When the patient is conscious of malaise, slight fever is present, the physical signs are few, consisting in slight dulness and a few crackling rales, cough is not marked until resolution begins, when there is expectoration of thin white mucus.

In the second form all signs are absent, only slight rise of temperature, marked prostration, but not easily distinguishable from the feebleness of old age. The patient dies suddenly, and is supposed to have been carried off by apoplexy till an autopsy discovers a consolidated lung.

In the third form, the onset is more sudden, the chill is absent, but fever is high, and may result in either delirium or stupor. The physical signs are more defined and the heart early becomes feeble. The case usually ends in death or insanity.

The special indications for treatment of pneumonia in children are based upon:

1st. The frequency with which the pneumonia is secondary to a simple bronchitis or to that accompanying an infectious disease.

2d. The special liability to pulmonary collapse. These indications are prominent inversely to the age of the patient.

3d. The readiness with which high temperature develops in children, and the great danger of such fever if persistent.

Every case of measles or pertusis or bronchitis in a young child must be considered as under the possible imminence of a broncho-pneumonia, and treated accordingly.

Owing to the special liability of pulmonary collapse, it is of the first importance to keep the respiratory movements

deep and vigorous.[13] It is undesirable to quiet the cough prematurely; indeed, if the child be too quiet, it should be excited to cough by crying. Jacobi and Wiederhofer advise using the interrupted current for brief moments to stimulate the diaphragm.

Opium is apt to be dangerous; it should be used only in minute doses, if the cough be harassing and other sedatives have failed. Antipyrin and phenacetin in small doses are admirable sedatives and preferable to opium, but should not be relied upon as antipyretics. An important consideration at the outset of a pneumonia is to avoid the vascular stimulation which will doubtless be required later if the disease persist. The classical treatment with vascular depressants, such as squills and ipecac, I will not dwell upon.

The complex aim of vascular and nervous sedation may be so well attained by antipyrin and phenacetin that the more old fashioned drugs will not be required. The formerly much used emetic is rarely required at the beginning, but where there is much oppression later on, a dose of ipecac or apomorphia is extremely useful.

[14] During the initial period of active hyperemia, the cold Priessintz compress around the chest is much to be recommended. The thinness of the walls of the child's chest greatly facilitates the transmission of cold to the engorged lung, and under the permanent application of the frequently renewed compress the blood vessels of the surface of the chest dilate and derive from the central circulation. The effect may be seen directly upon the lung in diminishing the hyperemia—thence pain, cough and nervous excitement—and the general temperature may be so lowered that more heroic hydrotherapeutic treatment will not be required.

If, however, symptoms of collapse are threatened and the temperature falls while the respiration and pulse rise, the child grows more pale and quiet and coughs less, the

cold compress should be exchanged for stimulating liniments, capsicum and turpentine suitably diluted, or an oil silk jacket should be worn in the intervals of these applications.

[15] If the suffocative symptoms grow more severe, the child should be put in a mustard bath or in a sheet wrung out of mustard water until the skin is reddened. Then if the symptoms persist, Gerhardt advises a warm bath with effusions of cold water.

Oxygen should be used as soon as there is respiratory distress and not deferred until there is commencing cyanosis, as recommended by some authors. It is utilized by tissue not yet consolidated or obstructed, and certainly does give great relief in many cases and possibly averts death.

In marked contrast to this group of cases and conditions when the leading indication is to provide for aeration, there is the second group where the prominent conditions are the hyperpyrexia and cerebral symptoms. In these cases the general toxemia predominates in importance over the local lesion. At a temperature of 104 or over, associated with nervous symptoms, there should be no fooling with antipyretic drugs, but recourse be had to the great antipyretic—cold—either in form of cold sponging, wet sheet or cold bath. This great remedy not only lowers temperature, but enables the nervous system to resist toxemia as well as possibly to interfere with the life process of poisonous microbes.

In both of the foregoing clinical groups, stimulants are generally required. [16] First among these is strychnin. This powerful drug, beside stimulating the heart and respiratory centres, stimulates, as Spitzska's experiments have shown, all nervous and muscular tissue and thus the muscular fibre in the bronchi, an action especially needed in children. Digitalis is a much more dubious remedy. Cadet de Gassicourt and other French authorities have considered it their main reliance, but its use has been superseded by

less dangerous and more efficient medication. The diffusible stimulants are frequently useful.

Alcohol in children is not so satisfactory as in adults, and while it has its place it should not be given too freely. It lowers an arterial tension already characteristically low and thus threatens heart paralysis. It easily disturbs the child's stomach, and also, if used early, conduces to convulsions. Dr. Daniels, in the large out-practice of the New York Infirmary for Children, tells me she has not for several years used alcohol in pneumonia of children. Of course, the general indications for heart stimulants exist in children as in adults, but it is to be remembered that the relative strength of the right heart in infancy renders mechanical heart failure less likely. It is more difficult to measure variations in the force and volume of the small pulse and heart beat, and the rapidity of pulse in children is of less significance than in an adult.

[17] Although the lesions of pneumonia in elderly people so greatly resemble those of children, their treatment differs considerably. The state of the organism attacked is different. The arterial tension is high owing to the rigidity of the arteries, and the heart muscle is frequently structurally weakened even where there be no valvular disease.

[18] Drugs which, like digitalis, strophanthus and caffein, increase the force of ventricular systole, are most valuable. Digitalis should be given only for about forty-eight hours without interruption, either in infusion or in granules of digitalin to accompany the strychnin. The diuretic effect of digitalis and caffein tends to meet the second indication offered by the frequent inadequacy of the kidneys in old age. To meet the same indications and to combat the toxemia, other diuretics are advisable. [19] In marked contrast with the case of children, the treatment of hyperpyrexia is rarely required, but occasionally the permanent [20] cold compress is most useful. Stimulants are constantly

required, but in contrast to the child alcohol holds the first place. The arterial tension needs to be lowered. For the same purpose, nitro-glycerin is invaluable. On the whole, in the child our eyes are to be fixed upon the lungs and in an old person upon the heart.

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## DISCUSSION.

Dr. F. C. Shattuck, of Boston:—I am reminded of a case I saw a number of years ago. The child of a medical friend, a man of large practice and great skill, was taken down with symptoms which suggested meningitis. He called in a professional friend and the child was thought to have typhoid fever. I saw the child on the ninth day and there was a beautiful, typical pneumonic consolidation. Now they had not overlooked it; it was late in coming on. I was reminded of that case by Dr. Withington speaking of the late localization which sometimes occurs, and this was as pronounced a case of the kind as I have seen.

Those cases are also interesting the course of which stamps them clearly as croupous pneumonia, but in which no lung consolidation can be detected at any time.

ARTICLE XXV.

# SERUMTHERAPY IN PNEUMONIA.

By WILLIAM H. SMITH, M.D.,
OF BOSTON.

READ JUNE 12, 1900.

# SERUMTHERAPY IN PNEUMONIA.

In 1892, Friedländer discovered in a case of croupous pneumonia, a capsule bacillus, which he considered to be the etiological factor in the production of this disease. In 1883 Talamon found in a consolidated lung a diplococcus, which he called the pneumococcus; but it was not until 1886 that Frænkel isolated the pneumococcus from a sufficient number of cases of acute croupous pneumonia, to show that it had an etiological bearing in the production of this disease. He was able to cultivate the organism and to study its methods of growth and its characteristics. Since this time numerous investigators have confirmed the observation of Frænkel—Netter, Wiechselbaum, and, more recently, Pearce at the Boston City Hospital, and Wright at the Massachusetts General Hospital; and from the data gathered it may be said that the etiological factor in the production of acute lobar pneumonia, in from 90 to 95 per cent. of the cases, is the pneumococcus. The organisms which take part in the production of broncho and lobular pneumonia are more varied, and to-day it is well recognized that any of the following organisms may produce a lobular or broncho pneumonia: the tubercle bacillus, the diphtheria bacillus, the bacillus mucosus capsulatus, the bacillus of bubonic plague, and the influenza bacillus, or the various forms of cocci:—streptococcus pyogenes, staphylococcus pyogenes, and the pneumococcus. One case is recorded where sarcinæ seemed to be the original invaders.

If, however, we eliminate the lobular or broncho pneumonias occurring in the course of the acute infectious dis-

eases and those secondary to an inflammatory process elsewhere, in a majority of cases the organism found, either alone or in association with other organisms, is the pneumococcus.

Since, therefore, in 90 to 95 per cent. of all cases of acute croupous pneumonia, and in a large number of the uncomplicated broncho or lobular pneumonias, the etiological factor is the pneumococcus, we shall, in our discussion of the serumtherapy in pneumonia, limit ourselves to the anti-pneumococcus serum. As soon as Frænkel's work upon the pneumococcus became recognized and the methods of isolation and cultivation of this organism became known, investigation by means of animal inoculation became rife. As early as 1886 Frænkel observed that rabbits inoculated with attenuated cultures of the pneumococcus withstood an otherwise fatal dose.

In pursuance of their investigations these observers used practically everything containing pneumococci. Attenuated cultures, glycerine extract of agar cultures heated to sixty degrees, heated bouillon cultures, filtrates and precipitates from cultures, dried organs of animals dying from pneumococcus infection, were used in these investigations, or infusions of the same in sterile water. Sputum, collected at the time of the crisis in cases of pneumonia, either fresh or after heating, was employed in these experiments; while the pleural exudate following croupous pneumonia was also used.

The results obtained from this line of investigation seemed to show that certain animals could, by the injection of pneumococcus cultures or exudates or secretions containing pneumococci, be rendered immune for a longer or a shorter time to an otherwise fatal dose of the pneumococcus. After this artificial immunity had been discovered, the next line of investigation was to determine if the serum of the immune animals was protective when inoculated into another animal; and, according to Mennes, Foa and Carbone were

the first to establish the fact that a mouse remained alive after inoculation with 2-4 drops of blood from an immune rabbit when this was followed by a fatal dose of pneumococcus culture. This is the first experiment in serumtherapy against the pneumococcus.

It remained for Emmerich and Fowitsky to show that the mouse remained alive if the immune serum was injected after the pneumococcus-culture—a fact also proved by the Klemperors.

The etiological factor in the production of pneumonia having been found, the fact that animals could be rendered immune for a longer or shorter time having been shown, and the immunizing property of such sera demonstrated when further inoculated into animals, it remained to discover the practical application of these facts.

At varying intervals there have been reported cases of pneumonia treated by the anti-pneumococcus serum. The Klemperors treated by subcutaneous injection a series of cases with apparent benefit, as shown by fall of temperature, slowing of pulse and respiration.

Lara reported the results in ten cases of pneumonia, the sera used being obtained from immunized dogs, rabbits, or glycerine extracts of organs of refractory animals. He considered the results encouraging.

In Naples in 1897, Prof. Pane reported that he had obtained anti-pneumococcus serum from a donkey and a cow that he had successfully used in treating a number of cases of pneumonia—twenty-three cases in all, with two deaths.

In 1898, before the Italian Medical Congress in Turin, Massalongo and Francheni reported upon Pane's anti-pneumococcus serum. They had used his serum in ten cases of the most severe type on alcoholic subjects with weak hearts and nephritis. They concluded that better results in this class of cases were obtained than if treated by the ordinary method.

De Renzi reported on a series of ten cases in 1898 treated by anti-pneumococcus serum. Severe cases were selected for the treatment. A cure resulted in every case.

Wiesbecker reported five cases treated by serum obtained from human subjects recovering from pneumonia. All five recovered, and there seemed to be a diminution of the subjective symptoms. The serum from blisters from pneumonia patients has also been used, but with little effect. Most recently Washbourne has succeeded in producing immunity in a pony after nine months' treatment with living and dead cultures, and has obtained a serum which seems to have powerful immunizing properties. The difficulty which all investigators had experienced up to this time was in their inability to keep the pneumococcus culture which they were using virulent. Washbourne applied the method of Pfeiffer of streaking agar cultures with sterile blood of rabbits, and by this means and keeping the blood at a temperature of 37.5 degrees C. it was found possible to maintain the virulence of the pneumococcus at a given level for a period of sixty days. The minimum fatal dose of the culture used by Washbourne was .000001 of a loop which held 5 m.g. The serum was mixed in varying amounts with a tenfold fatal dose of the culture and the mixture injected into the peritoneal cavity of rabbits, control experiments always being made with a minimum fatal dose. The smallest quantity of serum which would protect the animal under these conditions he considered equal to a unit. The strongest serum yet obtained from his pony equal to .03 c.c.—protected against a tenfold fatal dose. A c.c., therefore, equals 33 units. Sera from normal rabbits and guinea pigs would protect against the minimum fatal dose, but not against a tenfold fatal dose. Washbourne treated six cases and had no deaths. He considered that the serum should be inoculated as early as possible, and at least six hundred units should be injected twice a day.

Spurrel, Hartnett and Cook have reported cases treated by Washbourne's serum. Mennes has recently succeeded in obtaining a powerful immunizing serum from a horse. Neither Mennes nor Washbourne, however, wish to state positively that their serum is of value without further investigation. Certain subjective disturbances have been reported following these injections of the serum, such as excitement following the use of dog serum, agitation following the use of rabbit serum, and urticarial rash.

As the question stands to-day, it may be stated that, experimentally, animals may be rendered immune, that their sera has protective property, and that continued investigation is doing away with some of the difficulties which previously harassed the early investigators. But the fact still remains that in dealing with the pneumococcus we are dealing with an organism capable of many variations and degrees of virulence, and that the sera obtained under such conditions must of necessity vary both in strength and character. Washbourne's method of cultivation seems to be a distinct advance, inasmuch as he maintains life and virulence to the culture over a longer period than have other observers. His investigations are recent, and it is too early to state what the result will be. Experimentally, anti-pneumococcus serum seems to be of value. Practically, that it is of any great value does not at present seem to have been demonstrated, as the reported number of cases are too few; the series of any one observer too small. What the future of the serumtherapy in pneumonia will be it is too early to say.

ARTICLE XXVI.

# HYDROTHERAPY IN PNEUMONIA.

By SIMON BARUCH, M.D.,
OF NEW YORK CITY.

READ, BY INVITATION, JUNE 12, 1900.

# HYDROTHERAPY IN PNEUMONIA.

A RETROSPECTIVE glance at the treatment of acute diseases during the past half century reveals the fact that a revolution has taken place, which has culminated in the practical abolition of the treatment of these diseases. In our own country the revolution was initiated by Jacob Bigelow, a former member of this Society, by his work on the "Self Limitation of Diseases," published in 1835, and by his later book on "Nature in Disease." Reinforced by the good sense and captivating eloquence of your Oliver Wendell Holmes, American medical men have slowly, too slowly alas, changed from destructive to constructive therapy.

We realize to-day that, with few exceptions, the manifestations of disease are but the expression of pathological conditions, which no remedial agent is capable of removing. This proposition will not be construed by any fair mind as an abandonment of the patient to his fate, but simply to emphasize the fact that we no longer attack the disease by violent medication, as was formerly done by blood-letting and mercury and blisters, later by veratrum viride, and more recently by antipyretics. If the symptoms of disease were really the essence of disease, our complete control of the pulse by veratrum, of temperature by antipyrin, of sleep by chloral, of pain by morphia, of feebleness by the modern foods and stimulants, would to-day insure our triumph over disease. The fact that despite the response of the most significant symptoms to these powerful agents, patients continued to succumb, has awakened

the medical conscience to a realization of the truth of the doctrines taught by Bigelow, Holmes and Hooker, sixty or more years ago—that the patient rather than the disease must be treated.

In a malady like pneumonia, blood-letting and other spoliative agents relieved pain, softened the pulse, and lulled the doctor into a false security from which he was too often rudely awakened when heart failure presaged the approach of the end. So it is now with antipyretics, the chief value of which consists in enabling the patient to die with a more nearly normal temperature.

The old spoliative practice was abandoned when practical men discovered that it robbed them of the chief ally in the campaign against disease—the vis medicatrix naturæ.

If you reflect that for two thousand years the medical profession labored under the fatal error of fighting disease while the patient, being the battle ground, suffered from friend and foe alike, you will bear with me in paying a tribute of gratitude to the man who courageously blazed a pathway through the confusing mazes of spoliative medication to "Nature in Disease." I esteem it a high privilege to announce here, amid the very scenes of his professional activity, that like Jacob Bigelow I trust to "Nature" in pneumonia as in every other acute disease. Not blind as that of the Christian Scientist in prayer and faith, is my reliance on Nature, but ready in armed expectancy to intercede in her behalf when peculiarities of the patient or of the disease or conditions of environment point to a deviation from normal processes of restitution.

Isolation, rest, cleanliness, ventilation, food, drink, and if need be medication, should be so directed as to enhance the resisting powers of the patient—to so fortify him that his inherent powers have free play in eliminating the noxious products which threaten to overwhelm him.

Among these agencies I esteem Hydrotherapy as one of the most valuable. Water is not a curative agent for pneu-

monia, but its judicious use has afforded me much comfort and has doubtless contributed greatly to the diminution of mortality.

A somewhat active professional life of nearly forty years, spent in country, village, city, army, family and hospital practice, has brought me to the conviction that pneumonia is an infectious disease, which tends to destroy life by enfeebling the nervous and circulatory system, and that the indications for overcoming this lethal tendency are :—

1. To fortify the nervous system.

2. To sustain the heart, whose integrity is of vital import in overcoming the local lesion and in removing inflammatory products by a vigorous circulation.

3. To strive for elimination of noxious products arising from the life and death of the diplococcus.

4. To render the patient comfortable by reducing high temperature, deepening inspiration and producing sleep.

Flexibility is a notable quality of Hydrotherapy. By various procedures, changing of temperature and duration, we may adapt the application of water to the most opposite conditions for the fulfilment of these indications.

In no disease is this better exemplified than in pneumonia. Although its general manifestations resemble in many respects typhoid fever, its local manifestations differ from the latter as the Eberth bacillus differs from the diplococcus of Fraenkel. While in typhoid fever the cold bath, preferably as suggested by Brand, has proved of surpassing value, such a bath is not adapted to a patient suffering from pneumonia. The reason is clear at the bedside. The typhoid patient resists temperature abstraction with much tenacity ; the pneumonia patient, as a rule, quickly responds to heat abstraction. This clinical fact has often enabled me to confirm a tentative diagnosis, especially in children between eight and sixteen years of age.

While the typhoid patient, as a rule, bears the disturbance involved in tub bathing fairly well, such a procedure

is extremely distressing in pneumonia by reason of dyspnœa, cough and pain. Although Vogl of Munich, Folsom of Boston, and others, have reported good results from the cold full bath, I have abandoned them in adults for these reasons. In the pneumonia of young children, who are easily lifted, I still use full baths of moderate temperature (95°-80°), or affusions of water of lower temperature (70°-60°) in the broncho-pneumonias with obstruction, because pleurisy is usually absent, and the shallow breathing and deficient oxygenation due to bronchial obstruction are greatly relieved by the agitation, crying and coughing, incident to the full bath, with friction. It is my rule in all cases to be present during the first bath, in order to note the reaction and obtain other information for future guidance, because individuals differ materially in their response to bathing.

While each case demands special study with regard to baths, I usually begin with a tub bath (given alongside of the bed) of five degrees below the patient's temperature, and diminish the bath two or three degrees at each repetition in four hours until 80° are reached. The child's head and face are bathed in water at 65° before entering the bath, and gentle friction is made over the body during the entire bath. During the interval between the baths the method pursued in adults is adopted. In the latter, I have for the reasons stated substituted the wet thoracic compress for the full bath. It is my custom to have the rectal temperature taken every hour when the patient is not asleep. So long as the thermometer registers over 100°, a compress made of three folds of old coarse linen wrung out of water at 60° F. is wrapped around the chest from the clavicle to the umbilicus. It should be long enough to lap over one inch in front, and so slit in its axillary portion that it may rise easily up to the clavicle without leaving rough folds in the axilla. This compress is

smoothly wrapped around the chest and covered by one larger of thin flannel, an inch wider and longer.

The effect of such an application of cold is so readily observed that it would seem needless to dwell upon it, did not so much misapprehension exist in the average medical mind on the object of cold applications in febrile disorders, that it is necessary to again and again define it, to remove these erroneous impressions. Pardon me if I advert to some familiar physiological data. We know that cold and heat are thermic irritants which stimulate when mild, depress when more severe, and destroy vitality when sufficiently intense. Applied through the medium of water, and regulated with regard to temperature, duration and mechanical impact, we may so modify these thermic impressions as to produce therapeutic results for which we look in vain in other remedies.

The primary effect of cold is that of irritant to the sensory network of the skin. This has been called shock, an error which is made evident by the definition of shock. In Gould's Dictionary, shock is defined as "depression, a grave effect produced by severe injuries, operation and strong emotion." To produce a shock with cold water, it would be necessary to put the individual into a tub of water very much below the body temperature and sufficiently long to prevent reaction. If the water were near the freezing point and he could not escape, shock would ensue. The individual would become unconscious, and the vitality of the skin would be destroyed if he survived long enough to suffer from frost-bite. Bear in mind that the same effect would be produced by hot water under similar conditions, and yet no one ever speaks of the shock of hot water. The truth of the matter is—and I trust you will pardon my offering so simple a proposition—that cold stimulates when mild, and shocks when it is intense. Upon this principle we stimulate the nerve centres in pneumonia. A compress

containing water at 60° F. when applied around the chest of a patient having a temperature 40° higher, produces an irritation of the sensory terminals in the skin, which convey it to the central nervous system and thence reflect it upon the organs receiving impulses from the latter. This is plainly evidenced by the patient's momentary gasp, the heightened pulse tension, the deepened and prolonged inspiration and the lowering of temperature.

By changes in the preparation of the compress we may modify positively the effects aimed at. If, for instance, the body temperature is not very high, say from 100° to 102°, the compress may be more thoroughly wrung out, so as to make the impression of cold more brief, reaction more rapid and less enduring. If the temperature be high, 103° or above, the water temperature may be raised to 65°, more water may be allowed to remain in the compress, thus rendering the reaction more slow and enduring and abstracting more heat. In the first instance the application will be more stimulating, in the latter more soothing and antipyretic. If the patient is easily chilled or does not react readily whether the body temperature be moderate or very high, the compress may be allowed to remain longer without change, its repetition may vary from half an hour to an hour, or even longer, according to the patient's condition, his reaction and other effects produced and aimed at. Such cautious adaptation will gradually improve or regulate the reactive capacity of the patient, and sooner or later will enable him to bear more frequent repetition. It must always be borne in mind in all applications of cold water, that shock is to be avoided; there should be no prolonged chilliness, no chattering of teeth, no cyanosis of lips, nails or face; in fine, every manifestation which indicates a depressing effect demands a modification of the procedure or its abandonment if need be. And right here let me emphasize a much neglected point. We do not abandon quinine

in malarial fever when it produces distressing effects. On the contrary, bearing in mind the importance of the remedy, we circumvent its distressing effects by changing the method or time of administration, etc. Cold water is often entirely abandoned when it produces unfavorable manifestations, which is a serious error. Treat it as you would any other remedial agent; change the method, the temperature, the duration, and you will be gratified by the result, for it is a most flexible agent as I have shown.

Besides the general effect referred to, we observe a local stimulating effect upon the cutaneous circulation. The arterioles contract under the cold compress, but they quickly dilate again as is evident from absence or disappearance of chilliness and the gradual warming up of the part, so that when the compress is removed in an hour, it is quite warm. This dilation, be it remembered, however, is not a passive one like that under a warm poultice. The skin does not wrinkle and become cyanotic, but remains smooth and becomes ruddy. Moreover, repetition again stimulates the walls of the arterioles to contraction and dilation, propelling the blood flow through them, and thus relieving the overburdened heart of much labor. Romberg and Paessler have recently confirmed by laboratory experiments what I have several years ago and repeatedly since that time insisted upon, that in acute infectious diseases we encounter disturbances of the circulation which manifest themselves clinically as reduced tension and diminished filling of arteries and which are commonly described as heart failure. Undoubtedly this condition of the peripheral vessels bears a very large share in the production of cardiac inadequacy as I have sought to impress when explaining the rationale of cold application in typhoid fever. Romberg has shown by experiments with injections into rabbits of Fraenkel's diplococci that the circulation is damaged by a paralysis of the vasomotors while the heart itself remains unaffected.

By restoring the lost resiliency of the cutaneous vessels, the compresses tend to relieve the heart of much labor—a labor, too, which it vainly endeavors to compensate by increased pulsations—the sum of which too often leads to heart failure.

Upon this principle we endeavor to meet the second indication in pneumonia.

The third indication—the elimination of noxious products, is signally met by the improved condition of the central nervous system and of the heart, these presiding over all the functions of the body. The urine shows by its increase in quantity and toxicity that the most important noxæ are eliminated with great activity under the external application of cold. In addition to the latter the systematic administration of ice water (45° F.) four ounces every two hours, acts as a cold douche to the stomach and by increasing arterial tension increases urinary excretion. Allow me here to controvert an erroneous idea, which is almost universal, viz., that it is the increased quantity of water imbibed which increases the urine. (I have observed an increase of 100 to 400 per cent.) Glatz has shown, and I have confirmed his finding, that the imbibition of very hot or very cold water acts upon the arterial tension, precisely as its external application and not by reason of the quantity drunk. This is readily demonstrated by the fact that only a slight increase of urine is produced by drinking tepid water, and the quantity is always increased with decided differences between the body temperature and that of the water drunk. It is my custom therefore to alternate four ounces of milk or other liquid food with four to six ounces of very cold water (45°) each and every hour when the patient is awake. The result is systematic and moderate feeding and renal stimulation.

The fourth therapeutic indication is certainly fulfilled by the mild application of cold in pneumonia. The comfort of the

patient is greatly enhanced by the reduction of temperature, deepening of the inspiration and general bien aise which are renewed at each application. The cold compress when its temperature is equalized to that of the patient's body really becomes a poultice, soothing in its effect, relieving pain, dyspnœa, shallow and rapid breathing, but not relaxing the cutaneous vessels nor increasing body temperature like the poultice. It also increases leucocytosis, as has been established by Thayer, Winternitz and Rovighi.

To sum up briefly the effect of hydrotherapy in pneumonia I would say that its judicious application fulfils all the therapeutic indications in this disease by meeting all the depreciating conditions which lead to a fatal termination. Only one condition is unaffected by this treatment, viz., resolution. I have observed crisis only in about 25 per cent. of cases. The local course of the disease appears to be unchanged. Resolution proceeds slowly, but surely. The patient has a normal temperature, pulse and almost normal respiration from five to twenty days before all signs of consolidation have disappeared. In the meantime I permit him to go out in mild weather and endeavor to hasten convalescence by the usual measures, good food, gentle outdoor exercise, ventilation, etc. I am still unable to explain theoretically this retardation of resolution in 50 per cent. of the cases, except on the same principle as the relapses in typhoid fever after the Brand treatment may be explained, viz. :— there are more cases escaping death and therefore more cases to relapse. The cases which do not have crisis under hydrotherapy would probably be added to the great silent majority or pursue the same chronic course toward convalescence.

## DISCUSSION.

Dr. Z. B. Adams, of Framingham :—I did not come here with the intention of saying anything, but I expected, hoped, was encouraged almost to believe, that somebody would say something about bleeding in pneumonia. There has been no remedy mentioned since I came in having any special value in the treatment of pneumonia. There, gentlemen (holding up a lancet), is a little remedy which I always carry in my pocket which as a matter of fact will cure a case otherwise fatal of pneumonia at a certain stage of the disease. That, gentlemen, is a fact. You will say: "Why is it a fact? Explain it." I am not going into that. I say to you; *try it* and find out that it is a fact. That is the only way by which you can be convinced. Your minds are poisoned, prejudiced, against a remedy which, if used, saves our art from the charge of actually committing murder by its omission. This charge is well founded: it is a true charge. You can, *you can cut short, absolutely stop*, certain cases of croupous pneumonia at a moribund stage by the lancet; by bleeding and *bleeding freely*. I have seen it in many cases; but my authority is nothing. I assure you that this is known to skilled physicians. Now, then, what is to be done with a fact like that? Our theories, our science, seem to say it is impossible. What is to be done when you have fact on one side and theories on the other? Your theories must give way. The fact must have right of way, and it will. It is certain sometime or other to be acknowleged again, as it has been in the past. I want to show you where you have been placed by an unfortunate book; something you have read,—or your friends, your family, your patients, the world about you,—have read, has had a good deal to do with this. Two or three of the greatest novelists that have ever lived have been writing in this last half century—Englishmen. Among *these* I place, as having the strongest influence upon our social conditions, Dickens; and next to him Charles Reade. In 1861 or thereabouts Mr. Charles Reade wrote a novel of wonderful power, called "Hard Cash," and in that book he breaks out with reiterated charges of the most emphatic kind against the practice of bleeding. He treats it with a contempt for which words

seem scarcely strong enough. This book has exerted an influence upon the minds of men with regard to medical practice comparable in some sort to that of Mrs. Stowe's novel which we say precipitated the Civil War. The minds of men have been poisoned by that great novel, written by that man of genius, and the effect is still upon us. You may smile at this if you will. These things are deeper than you think. Your fathers and mothers, your acquaintances, your families, the community in which you live, give you your cue for practice; they have read this book, or at least have felt its influence.

DR. SHATTUCK:—You say at a certain stage.

DR. ADAMS:—I think it is not well to use bleeding after the seventh day. Let me draw a picture. I have been called in consultation and found a young, vigorous man, with pallor of the face, lividity of the lips and fingers, extreme dyspnœa, pulse so poor and rapid as to be scarcely perceptible. Distress very great and the eyes glassy with the expression of death. The face is covered with a cold sweat; there is an ominous rattle in the throat; the attempt to swallow liquids brings on suffocation. Doctors in consultation have declared that the case is hopeless, the patient being moribund. A vein is opened, but the blood seems too thick to flow. Now the vein is opened in the other arm and soon a full stream is flowing from both sides. The transformation which follows is most striking. Recovery is uninterrupted.

Let me not be misunderstood. Bleeding cannot be made a routine treatment. Do any pretend that there is such a thing in pneumonia?

Now this practice is approved by many physicians. It is not used because it has been ridiculed as "a solecism," outlawed as "a piece of barbarity." I ask you, is our art to be ruled by epithets? Here is a fine example of the fatal power of words, of "the cursedness of definition," to borrow John Hunter's phrase. In all clinical experience I will venture to say there is nothing more striking and also more indisputable, than the transformation produced in the moribund stage of lobar pneumonia by bleeding.

Hemorrhages, often profuse, occur in certain constitutions and in certain conditions of health; they are believed to be

salutary. Nature here sets us an example. Bleeding saves a life here which without it would undeniably be lost; and the most beneficent office of medicine is to save life.

Now see the converse of this:—That a robust patient, whose constitution is unbroken by excesses or disease, should be allowed to die in the early stage of croupous pneumonia *without being bled*, is, in my view, a stigma upon the medical profession of Massachusetts at the end of the 19th century. The mortuary return of "heart failure," justifiable on semeiotic grounds, does no credit to us in such a case.

I am glad to have had an opportunity to say this. I wanted to find an occasion to say it, for I believe it should be said.

Dr. Baruch.—I am very glad to hear the gentleman express his conviction, and I have every respect for his observations. I happened to be graduated on the threshold between the bleeders and the non-bleeders in 1862, having been taught to bleed, cup, blister, mercurialize and to spoliate the patient, in order to cure the disease. I industriously and religiously followed my teachers' precepts in the military hospitals of Richmond, where we had a good many cases of pneumonia. We used bleeding, veratrum and all these methods, and I saw many cases of pneumonia cured, as demonstrated by auscultation; and I saw the patient die from the debilitating effect of the treatment after the lung had begun to resolve. That was the turning-point of my practice in pneumonia. I ceased the spoliative method, and I have tried to follow Bigelow ever since, as far as I could, considering the treatment adopted by my confréres. I do not believe that the expectant treatment of pneumonia is correct. The expectant treatment is based upon the principle that you always expect something to happen which you are going to meet when it happens; that is to say, when the patient gets weak you begin to stimulate; when he has a great deal of pain give anodynes; when he does not sleep give hypnotics. Now I consider that treatment not a rational one. The proper treatment for pneumonia, for all infectious diseases, as I said, is to prepare the patient for these emergencies. Knowing as we do that pneumonia is practically a self-limited disease, as much so as is typhoid fever, whatever will enhance his resisting

capacity will assist in the patient's recovery. Whenever the treatment is adopted which I have dwelt upon this afternoon, viz., to regard always the condition of the patient and to prepare him for the worst as it were, not to meet indications after they arise, but prevent them, then I think we shall have results which will be very gratifying. I see a great many Italians of the lowest classes, a great many drunken Irishmen, suffering from pneumonia, in the Manhattan Hospital, and I have statistical evidence that we have diminished the mortality 40 per cent. among these terrible cases which almost invariably died under the former expectant treatment. I can cite case after case in which this effect has been produced by the treatment adopted, not by hydrotherapy alone, however, which is only a part of it. I want it to be understood that any treatment which will enhance the patient's strength is correct; even an occasional antipyretic I would not object to, because that reduces temperature and makes him comfortable. Hydrotherapy is not a curative agent, but simply a valuable agent to enhance the patient's resisting capacity to the disease, and whatever you can do to accomplish that is a true remedy.

The Chairman, DR. F. C. SHATTUCK.—I should not like Dr. Baruch to go away thinking that in our Boston hospitals we do nothing to try to prevent or forestall bad symptoms, waiting until our patients are nearly dead before we try to save them. The adjective "expectant" may be unfortunate. Perhaps "symptomatic" is better. The point is that we cannot at present treat the disease, but can only strive to enable our patients to overmaster it.

I will permit myself to make one or two observations. We have had this afternoon an interesting exposition of some of the more important aspects of a grave and common disease. Several things seem to me to have been brought into specially strong light. One of these is that the underlying causes of acute lung consolidation are various. Truly, curative treatment must, therefore, be various. For this two things are needful: In the first place we need some means of accurate, positive and ready clinical differentiation of the different forms of pneumonia. It is not enough to wait for a culture from the heart's blood and the different organs. The second essential is the discovery of an anti-

toxin, as Dr. Otis well said, for each form: —and two, or possibly more, of these forms may be coexistent. Until the above two postulates are met I do not believe we can do more with the treatment of pneumonia than we do at present. Until this knowledge is won we must perforce be contented, as Dr. Baruch said, to treat the patient and not the disease.

There is one point about pneumonia which I did not hear alluded to, and that is the fact that in certain cases the infection is very much more widespread than in the lungs. We define pneumonia as a general disease with a localization in the lung; and yet we see cases not so very infrequently where the whole system is full of the pneumococcus, as it were. I recall a case I saw a year or two ago which was fatal within forty-eight hours from the start, where at autopsy there were found pneumococcus meningitis, pneumococcus pneumonia, pericarditis and peritonitis. The treatment of a case of that kind, with sole reference to the lungs, cannot be of much avail. I must allude to that very interesting paper by Dr. Algernon Coolidge, Jr., and Dr. Townsend, some years ago, analyzing one thousand cases of pneumonia treated at the Massachusetts General Hospital from the time of its opening to 1890. Of course the first half was in the days of puking, purging and bleeding; but the last half was in the days of the expectant treatment, so-called: the mortality 25 per cent. in both. But that does not mean that the modern treatment is not any better. It seems to me it means that the modern treatment is a great deal better, because the material we have in the Massachusetts General Hospital to-day is a very different material from what was there in the first years of the hospital. Then this population was homogeneous and there were few poor people. The cases we get there now are of a different class, come in late, very hard drinkers often, subjected to every deleterious influence that a man or woman can be subjected to; the wonder is that any get well. And the fact that as many recover to-day as in the days of heroic treatment is evidence to me that the treatment is a good deal better. Personally I don't know any disease not necessarily fatal over which we have so little control as we have over pneumonia. That may be a pessimistic view to take. I hope it is.

ARTICLES XXVII.–XXXI.

# DISEASES OF JOINTS OFTEN DIAGNOSED AS RHEUMATISM, OR COMPLICATED WITH RHEUMATISM.

By HOMER GAGE, M.D., of Worcester.
CHARLES A. PORTER, M.D., of Boston.
ERNEST S. JACK, M.D., of Melrose.
SIDNEY A. LORD, M.D., of Boston.
WILLIAM H. PORTER, M.D., of New York City.

ARTICLE XXVII.

# TRAUMATIC JOINTS.

By HOMER GAGE, M.D.,
OF WORCESTER.

READ JUNE 13, 1900.

# TRAUMATIC JOINTS.

In his lectures on the Principles of Surgery, John Hunter, in discussing injuries to the joints, observes that "sprains are at least seldom perfectly cured," and while I think so strong a statement would hardly be accepted as true to-day, it must still be acknowledged that seemingly trivial accidents affecting the joints often result in very long and sometimes permanent disability; so that the saying, which must have been truer then than now, is still commonly heard, that a sprain is worse than a broken bone. Whenever the restoration of the function of the joint is long delayed or the pain persistent, the trouble is ordinarily ascribed to rheumatic complications. To what extent this is simply a convenient cloak to hide ignorance, or to what extent such complications do actually exist, it is the purpose of this discussion on diseases of the joints, as far as possible, to explain.

To introduce the subject, I wish very briefly to consider the effects of traumatism upon the joints, the changes which are commonly produced in the structures in and about the joints, how these changes explain in many instances the delay in the restoration of function, and what may be done to prevent such delays. For the purposes of this discussion, I wish to exclude all injuries which are complicated by fractures or dislocations, or by external lacerations, and to consider only those which come naturally under the head of sprains or contusions.

The effects of such injuries vary very much according to the amount of force used in producing them, and according

to the manner in which that force is applied. But there is one thing common to all injuries which are sufficient to cause a suspension of function, and that is, a laceration of some of the soft parts in or around the joint. It may be from direct violence, as in a bruise or contusion; it may be from indirect violence, as in the stretching caused by a twist or wrench; it may be complicated by synovial effusion. If severe, it may be manifested at once by the resulting discoloration, or the displacement of cellular structures may be manifest only upon microscopic examination. I believe, however, that as in concussion of the brain so in sprains and contusions of the joints, impaired function can be produced only by structural change. These changes may affect the cartilages, the ligaments, the synovial membranes, the muscles and their sheaths which lie in proximity to the affected joint or to the soft parts around and outside the joint—they may affect any or all of these structures. Injury to the cartilages may result in erosion and ulceration, must always be attended by injuries to the more superficial parts, and be slowly and imperfectly repaired. The ligaments, whether torn or stretched, will be thickened by the exudate which is deposited about the torn fibres—may contract adhesions to other structures, or be shortened or distorted by their resulting cicatrices. The synovial cavities may be obliterated by adhesions. Tenosynovitis may cause adhesions which prevent the play of the tendons in their sheaths, or the effusion of lymph in the soft parts about the joint may be so great as to interfere with its motion. The presence of blood clots in the tissues, with their subsequent organization, furnishes another element to favor the formation of adhesions and the contraction of resulting cicatrices. To a greater or less extent, these are the changes following every sprain or contusion, according to the severity of the injury. The important feature of them all is the tendency to the formation of adhesions and con-

tractions, and just so far as these are permitted to occur delayed and imperfect results will be found.

That treatment will be most successful which, while controlling exudation and permitting repair, provides at the same time for the maintenance of motion. This has been secured by various combinations of rest and compression, with both active and passive exercise. Fixation and compression are certainly the most efficient agents for the control of swelling and the relief of pain, and are, therefore, especially indicated at the outset. Compression also favors resolution, but does not prevent adhesions and contractions, and both are likely to follow too long-continued rest. A healthy joint may be held immovable for a very long time, and yet promptly regain its usefulness, but an injured joint would, during the process of repair, have acquired at least a partial anchylosis requiring weeks or even months to overcome, varying according to the extent of the damage. Rest and fixation should be continued only so long as is necessary for the relief of pain. They should give place first to the use of carefully applied massage—not indiscriminate rubbing—to stimulate the lymph currents and promote absorption of the effusion; and this should be followed, as fast as the disappearance of pain will permit, by some form of active or passive motion to maintain the elasticity of the tissues and prevent the formation of strong adhesions. In the milder cases this may be secured by the method of strapping, revived by Gibney a few years ago, which permits of limited voluntary exercise, and may often be used from the time of the very first dressing. In the severer cases, rest and compression must be maintained for a considerable time; but even then, after the first two or three days, must be supplemented by systematic passive exercise at frequent intervals, such exercises to be increased as fast as the subsidence of the pain will permit, and the amount of fixation diminished as rapidly as possible. It is well for

us to remember, I think, that the principle of rest, like many other good things, may be carried too far. It should give place to a carefully regulated and controlled use just as soon and just as far as the sensitiveness of the joint will reasonably allow. Keeping this always in mind, I believe that many of the delayed recoveries and imperfect results for which we have felt obliged to fall back upon the theory of some constitutional diathesis, may be satisfactorily avoided.

In the neglected cases in which swelling and stiffness have not disappeared, but, with pain and soreness, continue to interfere with the use of the joint, the problem is a different one. The effused blood and lymph have become partially organized, adhesions have been formed, and ligaments and tendons have already contracted. In these cases of chronic pain, rest relieves pain but favors anchylosis. Compression, combined with exercise, as in the method of strapping with adhesive plaster, is often useful, and sometimes sufficient. It is, however, especially in these cases that massage with involuntary as well as voluntary exercise, accomplishes most, and here we have much to learn from the violent methods of the bone setters and the osteopaths. If the anchylosis is thoroughly broken up under ether and the joint immobilized, the adhesions quickly reform. Some rest is necessary for the relief of the pain which follows the operation, but the massage and exercise, to be efficient, must be as vigorous as the patient can comfortably stand—much more vigorous, frequently applied, and persistent than in the case of freshly inflicted injury. A single instance from my own experience will, perhaps, best illustrate its necessity. A young man was thrown from a bicycle, injuring the elbow. No fracture was discovered at the time, and when he first presented himself to me, five weeks later, there was no swelling and no deformity, but almost complete anchylosis. He was advised to have it

vigorously rubbed and exercised and to use it as much as possible himself. Two or three weeks of this plan being followed by no relief, I etherized him, broke up the adhesions, found that there was no bony obstacle to the motions of the joint, and had the rubbings continued, but with no practical benefit. He was sent to a distinguished masseuse in Boston, who advised a continuance of active and passive motion, and assured him, as I had done, that good motion would ultimately be restored. At the end of a year, with the arm still anchylosed, he consulted a natural bonesetter, and at the end of a month had a useful arm, which has steadily improved until it is almost as good as it was before the accident. He tells me that the method of treatment by passive motion and rubbing was identical with what we had been doing, but that very much more force was used —that it hurt him a good deal at first, but after a few treatments the pain began to diminish and soon ceased altogether. This, and similar experiences, have taught me that where tolerably firm adhesions and contractions have occurred they can be overcome most quickly and most easily by the exhibition of a corresponding force in their manipulation, applied persistently, regularly, and at very short intervals.

Recently a new agent has been brought forward to assist in relieving these most obstinate cases, viz.: the hot air treatment, given most easily by the so-called Betz apparatus. It is practically baking the limb in a hot air oven in which the temperature is carried approximately to 300° F. From the careful observations and experiments in the use of hot air reported by Frazier in the Annals of Surgery for 1897, we learn that where a limb has been subjected to a temp. of 300° F. for an hour, a diffuse hyperemia of the integument may be plainly seen, there will be a temporary numbness of the part, and if there be not complete anchylosis, less pain and more freedom in the movements of the

joint. He believes that all the benefits are the results, not of any constitutional effects, but of purely local influences. These he finds chiefly in a locally stimulated circulation, which produces a temporary œdema, and permits of a certain amount of mechanical stretching, at the same time carrying away and flushing out the fluid that lies stagnant in the tissues. In other words, the hot air treatment accomplishes, perhaps, a little easier and more thoroughly, because the heat is greater, just what has been attempted by the old-fashioned method of the hot pack or the steaming in hot moist flannels. It prepares the joint, or puts it in a most favorable condition for massage and active exercises. To this extent it is a useful adjunct to the methods already described, but is of little avail without them.

In conclusion, I desire to express my belief—

1. That the injuries to joints, accompanied by loss of function, are always attended by more or less laceration of the tissues in or about the joint.

2. That the delays in the restoration of function are due in most instances not to any complicating diathesis, but to the changes incident to the repair of these lacerations and their effects.

3. That such delays are best avoided by an early resort to massage, and active or passive motion, and are favored by too long a continuance of rest and fixation. And

4. When such delays have occurred they are best overcome by more vigorous and persistent manipulation, supplemented by the application of heat or such other agents as may best stimulate the local circulation and favor the elasticity of the tissues.

ARTICLE XXVIII.

# SEPTIC AND GONORRHŒA JOINTS.

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# SEPTIC AND GONORRHŒA JOINTS.

THERE is probably no medical term which is made to cover so many sins and errors in diagnosis as the oft-used words "joint rheumatism." By dint of careful clinical study, and especially through the aid of bacteriology, certain types of joint disease have been gradually differentiated from this ancient chaos. Last to endure—it is to be hoped soon to go—is the rather indefinite phrase "gonorrhœal rheumatism." This paper deals with joint infections, septic and gonorrhœal, which may be confused with articular rheumatism. As this disease is probably also an infection manifesting itself chiefly in the joints, it is evident that we are dealing with more or less similar processes, which, though varying in etiology, must still have many symptoms in common.

No mention will be made of the septic infections which follow immediately upon operations or open wounds of joints, or of joint tuberculosis. There remain the infections which occur secondary to a suppurative process in other parts of the body, or following general infection through undiscoverable local lesions. The more carefully this question is investigated, the more we recognize that pathogenic organisms may enter the circulation even in conditions of apparent health. These organisms either die in the blood, are eliminated by the kidneys, etc., or localize themselves in various organs or tissues where they may be destroyed by the phagocytic cells or germicidal body fluids. If, through a general or local diminished resistance, the germs continue to grow, there results a general or local infection of

varying intensity. A favorite site for such manifestations are the joints or the adjacent epiphyseal ends of bones. Where a suppurative or septic process is known to coexist, errors of diagnosis are infrequent. In the infective diseases like scarlet fever, dysentery, pneumonia, meningitis, the attendant joint complications are usually recognized. Most difficulties occur in the cases of so-called idiopathic arthritis, where the mode of entrance of the organisms is undiscoverable, through the tonsils, pharynx, intestinal tract, a boil or pustule, a small healed wound. The germs which most commonly are found in such joints are the ordinary pyogenic staphylococcus, the streptococcus, the pneumococcus. Unusual infections occur from the typhoid bacillus, glanders bacillus, etc. Cases of gonorrhœal infection and the arthritis accompanying osteomyelitis or epiphysitis, are most frequently mistaken for rheumatism. In infants or young children often no existing cause can be found. In older children or young adults, exposure to wet or cold not infrequently precedes the joint inflammation, or so-called "rheumatism" sets in after an injury. There may have been a sore throat. The constitutional symptoms are in general more severe, the fever higher and less irregular, with a more rapid pulse. Sweats are uncommon, chills relatively frequent, the leucocytosis more marked. The pain is extreme and boring, limited in the earlier stages to the epiphyseal end of the adjacent bone; this sign soon giving place to general tenderness about the joint as it becomes involved. Of value for diagnosis is the definite interval by which the general symptoms and great pain precede the signs of local infection; thus differing from rheumatism. In superficial joints œdema soon appears and rapidly extends; redness appears later than in acute rheumatism, the joint capsule or periosteum may be perforated, when fluctuation becomes evident. Though more than one joint may be involved, such septic processes are usually

monarticular. Salicylates have little effect on the pain. As early diagnosis is all important to save the joint from disorganization, puncture, in doubtful cases, should be resorted to without hesitation. Not all septic joints have such an acute onset. For example, a boy eight years of age entered the Massachusetts General Hospital with the diagnosis of articular rheumatism of the right knee, duration five days. Salicylates had caused no improvement. Temperature 100, pulse 80; only moderate pain and tenderness. The effusion was slight; leucocytosis 11,000. The condition remained relatively the same for four days, when it was learned that he had fallen on a needle which had entered to one side of the patella. The joint was opened and found much disorganized. Cultures showed pure streptococcus infection.

Another boy of ten, without preceding illness, fell and struck his left knee. He was brought into the hospital three days afterwards with the diagnosis of acute rheumatism. There were high temperature and rapid pulse and several general symptoms. The joint was very tense and held immovably fixed. The skin was much reddened; pain and tenderness extreme. Under ether, three ounces of creamy, yellow pus were evacuated, which showed a pure culture of the pneumococcus. The subsequent course of this case illustrates the frequent burrowing of pus even after apparently thorough drainage. Several successive abscesses formed which extended between the muscle planes and fasciæ, and up and down the leg. From all of these pure cultures of the original organisms were obtained. Although in both of these cases at the time of operation it appeared as if permanent anchylosis would follow, at the end of three months the knees could be flexed to a right angle. With early operation in such cases the prognosis is probably not as bad as would be expected, especially in children and young adults. As gauze stimulates the formation of granu-

lation tissue, protective seems to be the ideal drainage material. Under it the synovial membrane is more quickly restored, adhesions are prevented rather than excited, irrigation and changing of dressings is almost painless, and the danger of pus backing up behind gauze wicks is not present.

To illustrate a joint infection with one of the rarer organisms, a case of Dr. C. B. Porter is of interest. A man of thirty-five entered the hospital with severe general symptoms and an arthritis of the great toe and knee. Cultures from the pus were sterile on ordinary media. Inoculation into guinea pigs, however, proved that the infecting organism was the glanders bacillus. There was nothing characteristic in the joint lesions.

When once the diagnosis of a septic joint has been made, the surgical indications for drainage and disinfection are so clear that there is no need to speak of their treatment.

The pathological processes in these septic joints are well recognized and need not be alluded to.

Of late several cases of joint infection with the typhoid bacillus in pure culture have been reported, and attention has been called to these typhoid joints, especially by Keen in his monograph.

Owing to the general condition of the patient the joint lesions are often overlooked. It appears to be characteristic that an insidious disorganization occurs in the articular structures which relatively early leads to dislocation. Indeed, it is this dislocation which in several cases has first attracted attention. Later, joint lesions may occur in typhoid fever, after apparent recovery, due either to the typhoid bacillus itself, or mixed infection with the ordinary pathogenic organisms which have entered through the healing ulcers. In such cases a subacute rheumatism may be simulated.

Though any of the above septic joints may occasionally be mistaken for rheumatism, gonorrhœal arthritis is a dis-

ease which has hitherto not been sufficiently recognized as quite different from ordinary rheumatism. Although continual advance has been made of late, particularly by the Germans, I do not believe that the majority of the profession yet realize how often a careful analysis of so-called "rheumatic cases" will lead to the discovery that the real nature of the arthritis is gonorrhœal. For many reasons the relative frequency of joint complications is hard to determine. Statistics from in or out-patient departments of hospitals and from private practice vary much, according to their source. The well known unreliability of venereal statistics, the great difficulty in diagnosing chronic or latent gonorrhœa in men, and especially in women; the comparative rareness of thorough bacteriological examination, with the frequent negative findings, even in undoubted cases,—all combine to make accurate statistics hard to obtain. One is often struck on carefully questioning gonorrhœal patients with the frequency with which mild or severe rheumatic symptoms have coexisted, and the more the physician's attention is directed to this subject, the more cases of gonorrhœal arthritis does he find. If, as seems probable, between two and five per cent. of the cases of gonorrhœa are complicated by arthritis, it is certainly not a rare disease. From another standpoint, although arthritis in connection with a gonorrhœal infection has been recognized for a long time, the causative relation of the gonococcus to it has only recently been demonstrated. The old idea, that the patient, run down by the disease, developed articular rheumatism, has been and is still advocated by some. While articular rheumatism may occur in such a patient, the nature of the arthritis, unless absolutely typical of rheumatism, should be regarded with much suspicion.

It has been demonstrated, also, that certain joint lesions occurring in connection with gonorrhœa have been due to the ordinary pyogenic organisms, though classed under

the general heading "gonorrhœal rheumatism." In such cases, where the bacteria have entered the blood from the urethra or elsewhere, the pyogenic organisms dominate the symptoms, and it is essentially a septic joint disease and not gonorrhœal arthritis. The gonococcus undoubtedly forms a powerful toxin, and certain writers have put forward the view that the joint manifestations may be due to this toxin, without actual localization of the organisms. Careful examination of the contents of undoubted gonorrhœal joints have so frequently proved sterile by smear and culture that there is some plausibility in favor of this toxic origin of the milder forms of arthritis. This subject will be spoken of later under Bacteriology. Until recently, gonorrhœa had been regarded as essentially a local disease, spreading by continuity. There are, however, a sufficient number of reliable reports which definitely establish that this organism may cause metastases in various distant parts of the body. Such localizations are numerous; in the joints, tendon sheaths and bursæ, pleura, peritoneum, meninges, sheaths of nerves, heart wall and valves, spleen, lungs, kidneys, periosteum, lymphatic glands, subcutaneous tissue, skin and parotid gland. From the circulating blood gonococci have been recovered several times, so that we have absolute proof that this organism may cause a general septicemia or pyemia, and may produce pus wherever localized.

As "grippe" with rheumatic pains in various parts of the body not infrequently complicates an acute gonorrhœa, or exacerbation of a chronic process, it is probable that mild systemic infection occurs, which never goes on to a definite arthritis. The rôle which gonotoxin absorption plays in producing these symptoms has not yet been determined.

### TIME OF OCCURRENCE OF METASTASES.

Though a period of from two to six weeks usually elapses before metastasis occurs, the joint complications may

first appear months or years after the original infection. Fingers' statistics seem to show that the joint lesions occur more frequently in chronic or relapsing gonorrhœa than during the acute stage; certainly in the older cases where few or no local signs exist, far more errors are made in diagnosis. Very rapid dissemination may occur within a few days of infection. For example, a case of Resniko's, a girl of twenty, four days after marriage, acquired an acute infection from her husband, who had purulent discharge at the time. She developed with the symptoms of general infection a severe polyarthritis. The ruptured hymen was regarded by him as the infection atrium. In chronic gonorrhœa the connection between an increase of local symptoms and a recrudescence in the joint is most striking and characteristic in many cases. The opposite may be true, and as in epididymitis, local symptoms may cease with the complication, or may show no change from the previous condition. Recent medical literature is full of articles emphasizing the long duration of gonorrhœa and demonstrating the difficulties of determining when infection has actually ceased. Particularly is this true in women, where the disease may appear to be chronic and mild from the start, and unknown to the patient. Owing to the numerous bacteria which exist in the vagina, culture methods and even staining are often most unsatisfactory. Negative findings after careful search are so commonly proved erroneous by the subsequent infection of another party that their value for exclusion is slight. In 226 cases of chronic cervical catarrh in prostitutes without symptoms, gonococci were found 117 times. In many of these cases the organism could be found only during or after menstruation.

Kopp reports 52 cases of chronic urethritis where repeated search was made for gonococci in vain; yet without renewed infection, recidives occurred in which the organisms

could be demonstrated. Therefore, in males also the latent period is often as impossible to diagnose, and is certainly far longer than is usually thought.

In a patient of mine who was positive that no new infection had occurred, gonococci were found in the seminal vesicles five years after the original disease. I dwell on this part of the subject simply to emphasize that, apart from the mere misstatements or ignorance of patients in regard to gonorrhœal infection, there are many cases where the physician can find no reason to suspect a connection between the present "rheumatism" and past venereal disease, and to show that the danger of gonorrhœal metastases endures for a long time. Though there is a general belief among genito-urinary surgeons that the metastases in males occur most frequently after posterior urethritis, it would appear that the depth to which the gonococci have penetrated into the mucous membrane is of more importance than the part of the urethra involved. A few days after infection the gonococci proliferate deep in the mucous membrane and are carried about within the migrating leucocytes. The not infrequent œdema or the prepuce and swelling of the inguinal glands are usually due to toxin absorption, though the presence of gonococci has been occasionally demonstrated. Though infection may thus occur through the lymphatic apparatus, it is believed that the organisms enter through the blood vessels.

Wertheim was able to show the gonococci in a thrombus of the bladder wall. The chain was thus made complete, and found to be similar to ordinary pyemic infection.

Though males are probably more frequently afflicted with joint complications than females, the disproportion is not as great as was once thought.

In babies, subsequent to gonorrhœal ophthalmia, there is naturally no disproportion between the sexes. In childhood cases occur in young girls from vulvo-vaginitis more fre-

quently than in boys of the same age. In adults, Nasse reports in one year forty-three cases, twenty-four in males and nineteen in females. Northrop, one hundred and seventeen in men and seventy-six in women. As attention has not been called sufficiently to gonorrhœal arthritis in women —and venereal disease is by them denied or unknown more commonly than by men—more cases have undoubtedly been overlooked.

Bedouin reports forty-two cases of arthritis in connection with the puerperium. While some of these were ordinary pyemic joints many of them showed the presence of gonococci. The lacerations attendant upon childbirth were considered as the source of entrance. The well known tendency of puerperal non-suppurative arthritis to prolonged stiffness or anchylosis is regarded by him as characteristic of a gonorrhœal joint. One case now in hospital illustrates this disease. Coincident with her confinement she had acute swelling of the metacarpo-phalangeal joint, ankle and knee on the right side. There was no evidence of uterine sepsis. The baby had gonorrhœal ophthalmia. Salicylates gave no relief to the pain; the local lesions, which will be spoken of later, were quite characteristic of gonorrhœa. The organisms were recovered from the joint contents in pure culture, as well as from the urethra.

In addition to the error of believing that women rarely suffer from gonorrhœal arthritis is the mistaken idea that such arthritis is essentially a monarticular affection, and different in that respect from acute articular rheumatism. Recent statistics have shown that more than one joint is involved in sixty per cent. of cases; yet, though several joints are involved, it *is* true that *one usually* bears the brunt of the infection.

The larger joints—the knee, ankle and elbow—suffer most frequently. The small joints of the hand, foot and fingers are more commonly attacked than in acute articular

rheumatism, though usually in connection with other joints. The jaw, spine and sterno-clavicular articulation are affected more often than in rheumatism. The sheaths of tendons, the bursæ, especially the bursa under the Achilles tendon, are favorite seats for gonorrhœal metastases. The tendons about a joint may be involved with it or independently. Trauma and over-exertion are predisposing causes. Curetting, the passage of sounds, too vigorous local treatment, may precipitate a metastasis or make an existing joint lesion worse. Exposure to cold or wet exercises no influence.

### SYMPTOMOLOGY AND COURSE.

In a disease like this, which varies in its manifestations from a slight effusion with little pain, to disorganization of a joint with formation of pus, accompanied by severe general and local symptoms, it is naturally difficult to classify the various forms and hard to describe any definite type.

Though at any given time the joint lesion may closely simulate acute or subacute articular rheumatism, it is, I think, possible in a great majority of cases to make a clinical diagnosis of gonorrhœal arthritis. In cases of acute joint disease, not conforming typically to articular rheumatism, a careful history will often make the physician at least suspicious of a past gonorrhœal infection. Thorough local examination may show the presence of an old urethritis or signs of an old gonorrhœa in women. Bacteriological examination of urethral shreds, discharges from the cervix, urethra, Bartholin's ducts or adjacent crypts, may show the presence of gonococci. When such evidence is wanting the course of the disease and the general local symptoms can alone be relied upon. The mode of onset varies. The majority of joint lesions are mild; particularly in connection with chronic gonorrhœa an acute or subacute synovitis develops most frequently in the knee. There is little pain, moderate stiffness, a varying amount of effusion

—which may rapidly disappear, or may persist with thickening of the capsule as a painless chronic relapsing synovitis. In such cases there is nothing characteristic, and unless in the acute stage the gonococcus can be demonstrated by puncture, diagnosis is extremely difficult. If one or more times this synovitis recurs with increase in the urethral signs, the diagnosis is probable. The cases which are most frequently confounded with articular rheumatism are more severe, beginning usually in the morning without preceding exposure to wet and cold, or tonsillitis. One of the larger joints begins to swell, with fleeting pain in several others. The amount of pain is so variable that from it little help can be obtained in diagnosis. It may be extreme, but is generally less than in articular rheumatism. The pain is usually worse at night. Several joints may be involved, or one joint may follow another in fairly rapid succession; but the tendency to improve in one as another becomes involved, so characteristic of articular rheumatism, rarely occurs in gonorrhœal arthritis. The joint most affected shows little tendency to improve, and remains swollen and tender for a long time, entirely out of proportion to the general symptoms. The amount of fever varies; rarely absent at the outset, it may disappear at the end of a few days, though the joint symptoms remain active. In severer cases it may last for a number of weeks, and soon assume the suppurative type with morning remissions. This is true in cases which have been proved to be uncontaminated by ordinary pyogenic organisms. Almost never does the fever curve show the markedly irregular remissions characteristic of real rheumatism. The acid sweats are wanting; endocarditis is very rare. The amount of effusion varies. On the whole it is rather slight, and the chief changes occur in the synovial membranes and periarticular tissues. In some cases the joint capsule becomes rapidly thickened without much œdema, but as a rule the subcuta-

neous infiltration is well marked, extending above and below the joint, and giving a doughy, boggy feel. The skin is not reddened, the papillæ are apparent, and the glossy, mottled blush characteristic of articular rheumatism is absent. The local heat and superficial sensitiveness are slight. In short, except for the tenderness and the acute onset, the gonorrhœal joint strongly suggests a tumor albus. For weeks the local signs are out of proportion to the general symptoms. Often the pain disappears within two weeks; yet for a long time tender points on deep pressure are found over the sides of the joint at the insertion of the ligaments or tendons. In acute cases, the joint is fixed. In the knee flexion occurs relatively early. The great sensitiveness to deep pressure, the early tendency to stiffness and anchylosis, are most characteristic. In the wrist, more than in any other joint, softened tender areas appear among the tendons, which suggest the formation of an abscess. Pus, however, is infrequent, and one finds only softened hemorrhagic granulation tissue. The contents of the joint may perforate the capsule, or infection by continuity occur. Residual abscesses, often unaccompanied by fever, may extend along the tendon sheaths or into the subcutaneous tissue.

The œdema and exudate about the joints and tendon sheaths persist longer than the serous infiltration of acute articular rheumatism, and after their disappearance the boggy thickening of the capsule and subsynovial tissue, with little effusion, is apparent; whereas, in rheumatism the joints usually recover in six weeks, gonorrhœal joints often remain stiff and swollen for two to six months. A non-traumatic arthritis, which persists for weeks in one joint, should always arouse suspicion.

In several cases I have seen at the hospital, especially in the wrist, acute tuberculosis was closely simulated. In such cases tuberculin may aid in the differential diagnosis.

Periarthritis of the shoulder, not infrequent in young adults, without the history of trauma, may be due to gonorrhœal infection.

### BACTERIOLOGY AND PATHOLOGY.

A great deal of work has been done on gonorrhœal joints in the past two years, especially by the Germans, as a result of which the behavior of the gonococcus in the joint and the resulting lesions are fairly well understood. Rindfleisch has published the results in thirty cases very carefully investigated by him. In nineteen, gonococci were demonstrated. In two, the staphylococcus aureus alone; in one, mixed with gonococci was found. In one, the staphylococcus albus alone, in another with the gonococcus. In nine cases no organisms could be found. Weiss has collected one hundred and twenty-one cases of undoubted gonorrhœal arthritis, from ninety-two of which the gonococcus was isolated. Young, of Johns Hopkins Hospital, recently reports thirteen cases of joint and tendon sheath infection, in which the gonococcus alone was present. The gonococcus apparently first localizes itself in the subendothelial tissue, there proliferates, and is carried into the joint cavity by the migrating leucocytes, or the desquamated endothelium. In the first few days the organisms are infrequent, in the joint fluid. At the end of three days the leucocytes and endothelial cells appear in about equal numbers, and the gonococci are most abundant, yet are conspicuous for their relative scarcity, occurring in twos and fours within and without the cells. The joint fluid is not a favorable medium for their growth. Degenerate forms soon appear, shown by their refusal to grow on culture, and the crowding of the leucocytes with more organisms. Subsequent proliferation occurs not in the joint fluid, but in the synovial membrane, as Rindfleisch and others have demonstrated. In one case, numerous organisms were present in the fluid on the third day, had disappeared by the

13th, yet could be demonstrated by culture and smears, in curettings from the synovial membranes.

In reviewing the cases reported of positive findings, it is striking that in all of them the joint infection was relatively fresh, or a recurrence had taken place. I can discover no mention of gonococci found in chronic synovitis. When we consider this early disappearance from the joint cavity, the relative scarcity of the organisms, and the well known difficulties in their successful cultivation, we can understand why so many aspirations may be sterile, and the necessity for early puncture of the joint for diagnostic purposes is evident. *Both* culture and staining methods should be used, for in many instances cultures have proved positive where no organisms could be found by staining, and vice versa. Though exudation, and even suppuration, has been produced by injection into joints of sterile products, gonotoxins, in view of the abovementioned facts it seems more likely that the gonococci have early disappeared from the joint, or failed of demonstration, than that local joint disease is caused by toxins absorbed from the urethra. In the effusion there is nothing characteristic. It is more apt to be blood stained and purulent than in acute articular rheumatism. König regards a reddish gummy, seropurulent fluid as significant.

The gonorrhœal process in joints is characterized chiefly by proliferative rather than destructive changes. The synovial membrane and periarticular tissues are much thickened and infiltrated, even at a distance from the joint; the granulation tissue formed is very richly vascularized. The capillary walls are thin, areas of hemorrhage are frequent, and blood usually escapes into the joint. In more chronic processes in the joint and about the tendon sheaths, the dark red, easily bleeding granulation tissue giving to the joint or sheath a velvety appearance, is suggestive of a gonorrhœal process. It is the rapid organization of this granu-

lation tissue and surrounding exudate which causes the early tendency to stiffness and anchylosis.

CASES.

A few cases which I have seen at the hospital will illustrate typical gonorrhœal joints.

A girl of thirty, who denied venereal history, entered the hospital suffering from debility. Three days after entrance, she suddenly developed, with moderate fever, an acute swelling of the right metacarpo-phalangeal joint and right knee. The thumb was swollen, tender, not red; moderate effusion in the joint. In the knee the synovitis was slight. The capsule was evidently thickened; periarticular œdema lacking. Pain severe, worse at night; leg, moderately flexed. The inflammation in the thumb quickly subsided, but the local tenderness in the knee steadily increased. Three weeks after the onset the joint was tapped, and a small amount of seropurulent fluid obtained, tinged with blood. This relieved the pain. Cultures were sterile on ordinary media. No organisms could be found by the microscope. As the fever continued, varying from 99 to 101½, and the joint had not improved, operation was decided upon. The knee was held moderately flexed, very tender on deep pressure over the external lateral ligaments, and presented the appearance of a chronic white swelling. At operation a small amount of turbid blood-tinged sero-pus escaped. The cavity of the joint was small, limited by the greatly swollen synovial membrane, which in places was an inch thick. Fibrin was small in amount. Already adhesions had formed between the patella, femur, and several parts of the capsule; the alar ligaments were greatly thickened. Cultures and smears were again negative. No organisms could be found in the sections from the synovial membrane. At operation a purulent vaginitis was found, but no gonococci could be demonstrated. A history of cystitis was finally obtained

four months before the present illness. The fever gradually subsided, but the joint swelling and tenderness persisted for several weeks with great stiffness, and ultimately anchylosis was absolute. Large doses of mercury and iodide had no effect.

Another case previously alluded to showed, coincident with confinement, a swelling of the thumb, ankle and knee. The symptoms in the hand and foot rapidly subsided, but in spite of salicylates the knee joint remained very painful, flexed to a right angle, not red, very tender at several points, with moderate effusion and moderate thickening of the capsule. This joint was opened by Dr. Harrington. The fluid was turbid, blood-stained, seropurulent. Examined under the microscope, the leucocytes were numerous. In a few of them diplococci which decolorized by Gram's stain were found; in two days pure cultures of the gonococcus were obtained by Dr. Wright.

A man of 42, who had contracted a gonorrhœa eight weeks before, entered the out-patient department with the following history: Two weeks ago, with moderate fever and much pain, he had swelling of the ankle, shoulder and wrist. In a few days the inflammation in the other joints subsided, but the wrist continued swollen. The hand and forearm were much swollen, the exudation firm and boggy; there was no redness, little heat. Motion at the wrist was impossible without great pain. In the anatomical snuff box and over the extensor tendons at the wrist, two apparently fluctuating areas existed. Gonococci were demonstrated in the urethra. On incision, the subcutaneous tissue was porky, the sheaths of the tendons were filled with a gummy, slightly hemorrhagic exudate. Communication with the joint could be made out. Gonococci were recovered from the pus and granulation tissue by Dr. Wright in smear and culture. The temperature was 100, the leucocytosis 26,000. Recovery followed very slowly, with marked tendency to anchylosis.

Another case presented the appearance of an ordinary palmar abscess over the head of the fifth metacarpal bone. Duration six days, with superficial redness of the skin and great tenderness at operation. No pus was found, but the adjacent joint was filled with seropurulent material in which were diplococci decolorizing by Gram's stain. No growth on ordinary media. The girl had contracted gonorrhœa four months before, and the organisms were still present in the urethra.

### TREATMENT.

Of the many drugs which have been advocated as beneficial in gonorrhœal arthritis, none in my experience has exerted much influence on the joint lesion. The pain is rarely relieved by the salicyl or coal-tar products, thus giving some aid in discriminating between a gonococcus infection and articular rheumatism. Morphia is often required in the acute stages. The treatment of the urethra should be begun at once, with mild, hot permanganate, or citrate of silver irrigation. No sounds should be passed during the acute joint disease. Laudanum fomentations, turpentine stupes, guiacol, or icthyol ointment, so often used, have appeared to me to be of little benefit. Most relief has been obtained from the continual use of ice bags, or dry heat, preferably with a hot oven, at a temperature of 250 to 400. The circulation is stimulated, pain relieved, and absorption of the exudate favored. It was hoped at one time that the temperature of the joint could be raised sufficiently to inhibit the growth of the gonococcus which is very susceptible to heat. This seems not to be the case. Flaming with the actual cautery is the best counter irritant and often relieves the pain.

Absolute immobilization by appropriate splints is the best treatment in the acute stage. As in other forms of arthritis, flexion of the knee may occur early. If gradual extension is too painful, an anæsthetic should be given at once,

and the joint straightened and immobilized, for if anchylosis results adhesions can be more safely broken by flexion than by extension. In the elbow the opposite is true, and this joint should be flexed to a right angle.

While the acute process usually subsides within two or three weeks, in many cases great pain follows attempts at motion, probably due to the rapid formation of adhesions within and about the joint. Here the physician is at a loss to decide upon the proper treatment. The joint grows stiffer day by day, yet movement is bitterly complained of by the patient. In such cases, advantage should be taken of the period of diminished sensitiveness to pain which usually follows the hot air bath. Massage should proceed movement. Unless the signs in the joints become worse, treatment should be firmly persisted in. At intervals, under an anæsthetic, the joint may be *once* fully flexed and extended, allowed to rest for a couple of days, when active and passive movements should be continued. In the acute stages, when the capsule is much distended, aspiration should be done for the purposes of diagnosis, and irrigation of the joint. In the subacute period the usually small amount of fluid is probably beneficial, separates the inflamed joint surfaces and prevents their adhesion. Pressure with immobilization should not be used at this time. The question of operation in gonorrhœal arthritis is coming more and more to the fore, and is advocated even in relatively mild cases by good surgeons, who report numerous cases where the fever, general symptoms and pain have been quickly relieved by operation, and earlier use of the joint followed.

By operation the toxins and gonococci in the joint fluid may be rapidly evacuated, and such treatment is indicated in all severe gonorrhœal joints, as in any septic arthritis. With proper precautions the danger from sepsis is probably slight in comparison with the advantage to be gained, good temporary results may follow operation in the knee, where

the surfaces can be seen, thoroughly cleaned, and superficially disinfected; but, as the gonococci will continue to proliferate in the synovial membrane beyond reach, the process, though checked, will often continue. As relatively few pure gonorrhœal infections lead to suppuration, the treatment by immobilization and early passive motion is safer for general adoption. Particularly is this true in the more complicated joints, as the elbow, ankle or wrist, where thorough irrigation and drainage are usually impossible, and where incisions among the adjacent tendons would not infrequently lead to the anchylosis which operation is intended to prevent. When pus can be demonstrated by fluctuation, œdema and redness, or aspiration of the joint or tendon sheaths, then only should operation be resorted to. Incision, irrigation, immediate suture, without drainage, should be attempted.

The passive hyperemia method, advocated by Behr, in the treatment of joint tuberculosis, has been used with good results in gonorrhœal arthritis. The limb is bandaged up to the joint; above it a flat tourniquet is applied, sufficiently tight to impede the venous circulation. The joint swells, the pain is relieved, and earlier motion is reported. This was true in two of my cases. In three others, no benefit followed.

## SUMMARY.

In this paper I have endeavored to show, first, that the gonococcus alone can cause arthritis, which, without the presence of other organisms, may be purulent.

Second, gonorrhœal arthritis occurs not only in acute gonorrhœa, but in the chronic or latent stages, which persist much longer than is commonly believed.

Third, the signs of such chronic infection in the male and female are often slight, and disregarded or overlooked by the patient and physician.

Fourth, in consequence, not a small proportion of cases are diagnosed as articular rheumatism, which are really of gonorrhœal origin.

ARTICLE XXIX.

# ACUTE ARTICULAR RHEUMATISM.

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READ JUNE 13, 1900.

## ACUTE ARTICULAR RHEUMATISM.
## DIAGNOSIS, PROGNOSIS AND TREATMENT.

THE clinical history of acute articular rheumatism is well known. Generally the onset is sudden, but sometimes the disease may by preceded by a mild degree of fever, indefinite pains in the joints, sore throat and especially tonsillitis. Chilliness, rather than any decided rigor, precedes a rapid rise in temperature, which is usually not very high, 102° to 104°, one or more joints become painful, and within twenty-four hours the disease is fully developed. Profuse perspiration, acid at first in reaction and of a peculiar sour odor, is diagnostic of this affection. The urine is diminished in quantity, of high color, and of high specific gravity. The tongue is coated, often there is thirst, and constipation as a rule exists. The joints become swollen, hot and reddened. Usually the larger joints are affected and any movement causes severe pain. If there is, in addition, evidence of any predisposition, hereditary or acquired, through any previous attack of rheumatism, the diagnosis would seem to be clear. And, it may be said, usually the diagnostic points are so obvious that we cannot fail to recognize the disease.

It is in those comparatively rare instances, when the disease is limited to a single joint, that the recognition becomes difficult. Anders[1] says: "The involvement of a single articulation (monarticular rheumatism) does sometimes occur;—the diagnosis of these cases offers great difficulties. On the other hand, he adds, an existing polyarticular rheumatism may become centered in a single joint and there

linger with great obstinacy." Of monarticular rheumatism Flint[2] says: "The chief difficulty in diagnosis, exclusive of the discrimination from gout, relates to cases in which the rheumatic affection is confined to a single joint. The problem in these cases is to distinguish rheumatism from a purely local affection of the joint, that is synovitis or simple acute arthritis. The following are the points in this differential diagnosis: The fixedness of the inflammation in a single joint from the first, in the latter, while in articular rheumatism it is rare that the mobility of the disease is not to some extent manifested. The existence of pyrexia in some cases of articular rheumatism prior to the affection of the joint. The want of correspondence between the degree of pyrexia and the local phenomena in rheumatic cases. The longer duration of the local affection in cases not rheumatic, and in certain cases the swelling and redness being greater than in rheumatic cases. The rapid disappearance of the local affection in rheumatism and the speedy recovery of the use of the affected joint, the disappearance and recovery being more slow in cases not rheumatic. The occurrence of endocarditis and pericarditis in a certain proportion of rheumatic cases."

Gonorrhœal arthritis, so-called gonorrhœal rheumatism, may affect several joints simultaneously. It does not migrate and often it is monarticular, affecting especially the knee. The local symptoms of pain and swelling are usually much more severe and, unlike true rheumatism, are more pronounced than the general symptoms. The pain and swelling is unaffected by the administration of the salicylates. The joint appears differently than in rheumatic cases. Loomis-Thompson[3] say: "In gonorrhœa the inflammation may extend beyond the joint, involving the tendon-sheath or, sometimes, the periosteum. In this manner the gonorrhœal joint acquires a somewhat different appearance from the acute rheumatic joint, the swelling of

the former being more fusiform, less circumscribed, extending for some distance beyond the joint and gradually tapering off."

Usually in gonorrhœa there is a history of a more or less recent infection. Gonorrhœal rheumatism is of long duration and may cause serious destruction of joint structures.

Osteomyelitis is confined to a single joint. The greater intensity of the local symptoms in this affection, the involvement of the epiphyses, and the more serious constitutional disturbances, are points to be remembered. It is especially important that we should consider carefully the diagnosis of this disease from acute articular rheumatism.

Osler[4] says: "The condition is, unfortunately, often mistaken, and, as the treatment is essentially surgical, the error is one which may cost the life of the patient."

Rheumatism in young children is more common than usually admitted in text-books. Often the symptoms, both local and general, are slight; but in these, as well as in the severer cases which must be differentiated from osteomyelitis, the early recognition is important on account of the cardiac complications that often accompany rheumatism and which may be easily overlooked.

The most important of the affections, giving rise to pain in several joints at the same time, to be diagnosed in pyemia. The joint symptoms in pyemia are usually preceded by fever for a longer time than in rheumatism. In pyemia the prostration is greater. There are rigors followed by perspiration. The perspiration is not acid as in rheumatism. The joint pains do not migrate. In pyemia there is the history, oftentimes, of trauma or of an abscess, and suppurative processes are early set up in other organs.

Gout attacks the smaller joints, the metatarsal joint of the great toe being a favorite location. The swelling and redness is more marked than in acute rheumatism. Rheumatism occurs in youth; gout is a disease of age. Rheumatism

occurs in those poorly nourished; in gout the reverse condition generally is true. Uric acid is found in the blood in gout; in rheumatism it is not. In rheumatism we have profuse acid perspiration; in gout it is absent. I have omitted the consideration of tuberculosis, of the joint manifestations of such infectious diseases as scarlet fever, and of acute exacerbations of rheumatoid arthritis, as they are preceded by symptoms characteristic of these affections, and should offer no difficulty in diagnosis.

The prognosis, aside from heart complications and those rare occurrences of hyperpyrexia and cerebral rheumatism, is good. The mortality, according to Loomis and Thompson, is from 1% to 2%, while Whittaker states that it is about 3%. The causes of death, then, are to be found in the complications so-called; in pericarditis and pneumonia; in the rare condition of hyperpyrexia and in those cases of cerebral rheumatism.

Many authors make no distinction between cerebral complications attending hyperpyrexia and cerebral rheumatism. Morgan[5] claims that cerebral rheumatism is not due to effects of high temperature upon the brain, and that rheumatism may occur without hyperpyrexia as the result of the special action upon the brain of the toxins of rheumatism. Aside from these cases of hyperpyrexia and cerebral rheumatism, which have a very grave prognosis, the mortality is greatest in pneumonia, which fortunately is a rare complication. Serious in itself, it is doubly so, associated, as it usually is, with endo- or peri-carditis.

Endocarditis oftentimes is recovered from; again it may be the beginning of serious valvular lesions to develop in years, particularly should there be recurrent attacks of rheumatism.

Ulcerative endocarditis and the possibility of embolism are to be considered. Pleurisy and bronchitis are less serious complications. Two cases have impressed me in my limited experience.

The first, a case of chorea in a child of ten, followed by acute articular rheumatism, cardiac trouble and death. Second, a case of tonsillitis followed by rheumatism, endocarditis and permanent lesion of the heart.

Through laboratory investigation, aside from surgery, we have made rapid strides in medicine, especially preventive medicine. Should we treat our cases of chorea and tonsillitis, particularly those cases having any rheumatic predisposition, by rheumatic remedies, and thus seek early whatever protection such treatment may afford against such complications as in the cases cited? Jacobi[6] says: "There are those cases in which chorea is not the final development of rheumatism and rheumatic endocarditis, but the very beginning of the disease and then referable to a rheumatic affection of the spinal membranes."

I have had no experience with hyperpyrexia or cerebral rheumatism, and, under the administration of the salicylates in large doses, these complications, I believe, are rare. Treatment embraces the administration of alkalies, of the salicylates, or a combination of both.

For treatment I employ the salicylate of sodium combined with pepsin and codeia. The pepsin has seemed to me to increase the tolerance of the salicylates; whether through any particular virtue in the drug itself or by the power of suggestion, I do not know.

The codeia quiets the restlessness, induces sleep and makes more endurable a condition far from enviable.

Lochbœhler[7] says of codeia: "There is very little or no danger of drug habit, its effects are sedative and hypnotic; it seems to have a special influence upon inflammation of serous membranes, and in moderate doses constipation, diminution of the urine and of the perspiration are not usually produced. In all cases of mania where anxiety, precordial distress and insomnia were present, it was found very useful."

Permanent injury to the joint rarely occurs in acute articular rheumatism. The reverse, however, is often true in what are termed those pseudo-rheumatic affections mentioned under diagnosis. Here the tendency is to chronicity, to suppuration and the destruction of the joint structures. What surgery has accomplished in such diseases as intestinal obstruction where early exploration is now done, applies to the treatment of all joint inflammations which fail to yield to the salicylates. J. O'Connor reports two cases of surgical treatment of acute articular rheumatism. The fact, that in these cases the salicylate of sodium seemed to be of no avail, although given in large doses, would suggest that possibly these were not cases of true rheumatism.

To the acutely inflamed joint there is much that can be done in the way of local applications. Authorities differ widely as to the importance of local treatment, some regarding it as futile, while others claim that it is most important. My own practice has been to wrap the joint in cotton batting, to support it if necessary by the application of a light splint, to bolster the patient in any position contributing to his ease. More active treatment seems uncalled for. Rest is of first importance, and no other measures are to be compared with it.

The number of liniments, evaporating lotions and soothing ointments recommended is without limit.

A useful and favorite application is Fuller's Lotion. Ichthyol is useful. Edlefsen[7] says: "With the application of ichthyol vasogen (10%) in monarticular rheumatism, particularly in recent disease, good results are obtained. In the polyarticular variety there is marked lessening of the pain and increased mobility of the affected joints." Osler speaks highly of the light application of the Paquelin thermocautery. The application of the oil of wintergreen is recommended. It is claimed that the application of an ointment containing salicylic acid and made up as follows:

salicylic acid, lanolin, turpentine, of each 5, lard 40, has been used with decided benefit, the acid being in evidence in the urine within a few hours. Allbut says: "I have seen no evidence of the absorption of salicylic acid applied in this way, nor any benefit to the patient. The local treatment by cloths wrung out in a solution of methyl-salicylate has many advocates who claim that it gives great relief." Morgan says: "In the New York Hospital in nearly all cases internal medication has been supplemented by the local application of methyl-salicylate, which has invariably given excellent results in rapidly relieving pain in the affected joints. Many cases in which gastric irritability contraindicated treatment by the mouth have quickly resulted in recovery when such local use of the drug alone was employed." In Germany ice is applied, but not to any extent in this country. Hot air applies only to chronic cases. The local injection of 10 to 15 minims of a 1% solution of carbolic acid has been tried.

In conclusion let me add that, when stiffness and tenderness persist about a joint, much relief may be obtained from properly directed passive movements and rubbing.

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ARTICLE XXX.

# NEUROPATHIC JOINTS.

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# NEUROPATHIC JOINTS.

It is obvious that the term "neuropathic joint" might reasonably be of very wide or extremely limited application, according to whether we had most in mind the fundamental rôle played by the nervous system in all vital processes on the one hand, or on the other the smallness of the number of joint affections generally regarded as essentially trophic in nature.

I shall apply the term in this paper to all those affections which seem, from the evidence at present available, to owe their characteristics to the direct influence of the nervous system, whether strictly trophic or not. The limitations of time will prevent any more than passing mention of the neuro-mimetic and so-called hysterical joints, which indeed do not fall within my province, for they depend upon disorder of ideas, are psychopathic in origin, not resulting from physical lesion. They have moreover but slight bearing on the differential diagnosis of the profoundly altered joints with which this paper principally deals.

Taking up first the classic examples of true nervous arthropathies as they are called—those of tabes dorsalis and syringomyelia—we find that the anatomical characters are almost identical in the joints, notwithstanding the great and essential differences shown by the diseases as a whole, so that much of what can be said of one applies to both.

It is of primary importance to recognize the facts that the joint lesions of these two cord diseases are in typical form almost absolutely limited to them, that they occur with frequency, 5% to 10% of all tabetics and about 10%

of syringomyelics being thus affected, and that conspicuous among the symptoms in both troubles are extensive losses of sensation. The course of these arthropathies need not be here described in great detail.

The following mode of onset is not at all uncommon. Entirely without warning, entirely without pain, entirely without evident cause, during some mild muscular action, absolutely without effort, often quite before any development of ataxia, the leg gives way or the arm drops, and the joint—knee or shoulder, *e. g.*—becomes at once useless; or if not at once, in a few minutes or hours. For there quickly ensues an enormous, tense, fluctuant swelling, utterly destroying action of the joint. In its suddenness and extent this swelling is quite out of the domain of all other conditions besides tabes or syringomyelia, except that following the great crushing violence of accident cases. The swelling extends usually beyond the joint limits—this is very characteristic; in fact it is not uncommon for the whole limb, or one segment, to increase enormously in size with astonishing rapidity. The cause of this is a remarkable subcutaneous and muscular infiltration, hard, non-pitting, often visibly hemorrhagic. There is no pain, and signs of inflammation are absent.

The further course of these arthropathies is the reverse of the onset: absorption is slow, and rarely complete, there remaining permanently moderate hydrops, with eventual (sometimes extremely rapid) development of flail-joint or "*schlotter-gelenk*," a unique condition of painless, fully mobile luxation. This state of abnormal mobility does not necessarily require a long time for its production—it may often be detected to slight degree in the course of a very few weeks, or as soon as the swelling has considerably receded. During the stage of active distension it is not usually present. The extreme grades of it, resulting in those fantastic shapes which as Marie[1] says "shame the

double jointed man" and result even in perforation, are only found in chronic cases. After this stage there sometimes occurs complete *restitutio ad integram*, not without tendency to relapse however. There commonly remains grating on movement, and the neighborhood of the joint slowly enlarges. Termination in anchylosis is rare. (Schlesinger[2] reports two cases in syringomyelia.) Other than this, cure is not to be expected.

The pathologic anatomy of these lesions is most interesting. It resembles much that in arthritis deformans, and consists in rarefication and hypertrophy, of bone, cartilage, ligament and synovial membrane. Combinations of these changes are most diverse, and explain the clinical variations in large degree. Bony frangibility and weakness of ligaments are of especial importance.

There are two principal types of change—atrophy and hypertrophy. In the atrophic form there is sometimes immense absorption of bone, which may include a considerable part of the shaft,—of the humerus or femur, for example, —the heads of the bones having completely disappeared. The opposing bony joint surface is likely to be somewhat hypertrophied under the influence of abnormal attrition. The hip and shoulder usually show the atrophic form.

The hypertrophic form is more common; there are irregular bony projections, perhaps free pieces of bone, large and small, in the joint, the cavity of which looks shrunken, through the great development of connective tissue. In the middle of the joint the cartilage has disappeared. The fluid in the joint is thick, yellow, sometimes hemorrhagic, occasionally almost pure blood. The ligaments are destroyed and ossified in varying degrees. The synovial membrane is often luxuriantly fringed. Fracture into the joint is common. This last statement indicates that there is an extreme degree of bony fragility; in arthritis deformans such fracture, at least of the whole head of the bone,

as is here seen, is absent or extremely rare. Anatomically still other distinctions between the two conditions have been described.

What is the immediate cause of these arthropathies? In discussing this it will be important to mention the chief clinical variations. It is vital to point out that, in its etiology, it is impossible to separate fracture from the arthropathies themselves. There can be no doubt of very frequent coincidence of fracture with arthropathy; and in the joints above discussed, with large swellings, the relation is probably nearly constant. I believe it highly probable that, in such cases, the fluid is, much more commonly than at present supposed, purely the result of active hemorrhage. Brissaud's[3] interesting case illustrates this. Tapping is unfortunately rarely done, so that we cannot be sure.

It seems to me that this factor of hemorrhage is necessary to complete the theories of Paget, Volkmann, Kolisko[4] and others who believed that the whole cause of arthropathy lay in violent ataxic movements, sufficient to fracture bones and to rupture the joint structures. The great force of these unfelt, incoördinated movements, the incoördination being augmented sometimes by irregular muscular atrophy, must be acknowledged, and the potency of this cause admitted, in view of the occurrence of so-called spontaneous fracture of even thick and sclerotic tabetic bones (Kolisko). It is easily seen that joint hemorrhage would be highly apt to result from such violence, would be rendered even more likely by the erosion of soft structures, and would explain particularly well the rapidity of swelling. There is, further, special ground to suspect hemorrhage when, as very rarely happens, much pain exists (Brissaud).

But ataxia, if a factor in producing these arthropathies, is not their exclusive cause. For, how would it account

for their appearance in syringomyelia in which there is no ataxia, or in tabes before the development of ataxia, or for their following exceedingly slight direct blows? Or, how would it explain the enormously diffused serous infiltration (not explainable mechanically), or the progression of symptoms during quietude, and the continuance of bone atrophy when the articular surfaces are no longer in contact?

It is evident that the final cause of these arthropathies is other than the immediate mechanical influence of ataxia, and that a more subtle pathology is demanded.

Arthritis deformans, gout and syphilis, all of which have been widely believed in, and strenuously put forward as exclusive causes, will, it is obvious, not begin to account for the extraordinary phenomena just described, nor will they account for the milder forms of arthropathy which their manifestations much more resemble, for those milder forms pass finally into severe ones. (Strumpell[5], formerly skeptical, now admits that it is hard to avoid the assumption of trophic disturbance.)

The association of these arthropathies with tabes and syringomyelia in some perfectly definite way is quite evident —their rarity in other cord diseases points to this—likewise the large number of other phenomena of undoubted trophic origin in tabes and syringomyelia: it seems in fact quite necessary to assume a special trophic disturbance, to account for the joint disease in question.

Is this trophic disorder due to neuritis? There is much evidence in favor of this, but not enough to warrant the assumption that neuritis is the only cause. Its presence is not constant, though very frequent, as Pitres and Carrière[6] have shown. We do not know whether it is secondary to the joint trouble, or whether it is due to the cord lesion. It is most intense in the neighborhood of the affected joint, and this points to the first possibility as the true one.

The following may be regarded as a conservative view. Several factors undoubtedly can and do exist in the etiology of both high and low grades of the arthropathies; but the only absolutely constant element is the nervous system, which acts directly by influencing nutrition, and indirectly by causing ataxia. These factors are, in the order of their probable importance, central and neuritic trophic disorder; anæsthesia of the joints; traumatism: direct, and (through ataxia), indirect; muscular hypotonia; arthritic tendency and syphilis. They act probably in this way. The nervous trouble predisposes the joints to disease through alteration of the sensory neurons. That the sensory nerves, while furnishing sensibility to the bones and joints, in some way take care of their nutrition, may be regarded as proved. Now there is equal reason to suppose that normal synergetic muscle-actions depend on continuous afflux of stimuli from the periphery. Therefore, any interruption of function of the centripetal joint-nerves will result in incoördination, with consequent loss of normal support to the joint. This loss entails uneven and violent stretchings and pressures on the ligamentous structures and articular surfaces. The disorder increases in a vicious circle on account of the relation between muscle and ligament. Osseous hyperplasia or atrophy occurs, this being determined probably by increase or loss of blood supply from vaso-motor change. Irritation from rubbing is part of the cause of the exostoses. Latent arthritic tendencies, which, of course, must often be present, are hastened into activity, and pursue a more extended course than they would otherwise take. Continued use of the joint, on account of the absence of all pain, after the lesion has begun, is a most important element in causing further damage. It must be added that the early incoördination which occurs on the development of joint anæsthesia probably begins at once to damage the joint even before ataxia has been manifest.

This helps to explain the excessively early appearance of some of these lesions. The rôle of hypotonia etiologically is likewise constant, if less important. That of syphilis may be deemed comparatively unimportant on account of the fact that there is nothing to show that the disease is frequently present.

In concluding this matter of etiology, the close and interesting correspondence of the arthropathies to the seat of the chief cord lesion must be mentioned—localization in upper limb in syringomyelia, in the lower in tabes. On the contrary, when the gliosis is of the lumbar type the arthropathies affect the lower limbs. Cervical tabes in like manner has been associated with upper limb arthropathy. It is to be presumed that shoulder affections of tabes are always to be explained on the anatomical basis of cervical involvement, and that, in general, leg arthropathies occur the earlier in tabes.

The other joints affected in tabes are, beside the vertebræ, nearly all the articulations, knee, hip, shoulder and elbow being the most frequently involved.

The "tabetic foot" is of rather different type, showing constant tendency to hypertrophy of bone, including even tibia and fibula, and ending in anchylosis. Its sudden and painless beginning results from rarefication and crumbling of tarsal and metatarsal bones. The shape is unlike anything else; the foot looks shortened, thickened and rounded, and the vault has disappeared. The appearance known as Chinese foot is largely due to muscular atrophy (Senator[7]), and occurs in various other diseases. It must be distinguished from true tabetic foot.

In syringomyelia, the arthropathies of the small hand-joints and the severe grades of kypho-scoliosis, all ascribed to trophic disorder, are worth mention. The purity of the type in the hand is obscured by infection and suppuration (Schlesinger).

There are certain unusual events in the course of classic arthropathies. Notably the very rare occurrence of pain, sometimes severe, ascribed by Brissaud wholly to stretching from effusion. It is, however, not wholly understood. It is possible that the usual rupture of the capsule, allowing escape of fluid and permeation of the periarticular tissues, here fails to occur. It is not clear why pain appears unless we assume analgesia of the joints but not of the skin. Investigation of possible association with neutritis would be of interest. The presence of pain has been adduced without justification, in my opinion, as an argument against the causative influence of analgesia. Suppuration and inflammation are most uncommon and certainly obscure the type of tabetic joint when present. The pathology is obviously different from usual. Suppuration is almost invariably secondary to trauma, occurs in the late cases, and should not confuse. Its course is remarkably mild, and in general the reaction of these joints to infection is slight.

The diagnosis of these joint lesions is rarely difficult except in certain early cases not long under observation. Mistakes would almost never occur if the possibility of cord lesion were in mind. Enough has been said to show the distinctions from simple fracture and luxation. There is, however, a condition of habitual luxation of the shoulder joint in syringomyelia, often preceded by direct trauma, which is of great practical and medico-legal importance. This may occur before obviously extensive change in the joint, and the patients have usually come to the surgeon unaware of central trouble. Schrader[3] reports fifteen cases. Some of these had had previously no cord symptoms.

Occasionally an early case with slow, chiefly hypertrophic changes, especially if tending towards anchylosis, will be confused with osteo-arthritis. The resemblance may be close clinically, but these cases are uncommon and rarely

long unassociated with symptoms of the central lesion. There may be a history of undue mobility. Characteristic involvement of small joints is lacking. There is also likely to be much more extensive periarticular and subcutaneous ossification than occurs in arthritis deformans, and a greater liability to the presence of large, free, bony masses in the joint.

In those cases in which, like that of Dercum and Spiller[9] (syr.), grating in the joint precedes by years the development of typical arthropathy, the diagnosis may at first be impossible.

The extreme atrophic cases with luxations are unmistakable.

There have been reported anomalous combinations of symptoms resembling both syringomyelia and tabes; the greater tendency in the former to diffuse ossification in the limb and to necrosis of bone would assist in the diagnosis of any joint trouble.

The complex of Morvan's Disease, so-called, is common to both leprosy and syringomyelia, and need not be further considered as such. Leprosy occasions difficulty only in hand affections and only in atypical instances.

One case of central cord tuberculosis produced typical tabetiform arthropathy and was not diagnosed *intra vitam* (Schlesinger).

Hematomyelia, a condition of hemorrhage into the cord in which the distribution of the blood is much like that of the gliosis process, appearing generally after accidents, may be in question. One doubtful case of this kind, that of Goldthwaite [10], has come to my notice.

Syphilis must always be considered as a remote possibility, simulating perhaps more the foot affection than those of the other joints. Practically speaking, antispecific treatment never benefits these troubles. When it does do so it might be expected that other luetic symptoms would

be present; but this is not true, otherwise a suspicion would be warrantable that not tabes but spinal lues were the central lesion.

Londe [11] calls attention to one case of true arthropathy in progressive muscular atrophy of the spinal type, and to one in chorea. He draws a fundamental distinction between the genuine lesions occurring in those diseases and gliosis and tabes, on the one hand, and all other nervous joint disorders on the other; asserting that, in the latter, one of two factors—infection or autointoxication—is always necessary for the production of the arthropathy, plus the nervous element. The nervous system is supposed to determine principally the location of the joint trouble in such cases, and Londe is right on the whole in thus assigning to it a subordinate part. I think, however, that two additions may be made to his list of diseases in which genuine arthropathies occur, to wit, central tuberculosis of the cord and certain instances of the trophic variety of intermittent hydrops, and perhaps hematomyelia.

Those non-genuine arthropathies emphasized by Londe are seen most frequently and typically in myelitis, neuritis and hemiplegia, also have been reported in Friedreich's disease, ataxic paraplegia, bulbar paralysis, and tuberculosis of the spinal column. They are all rare, and occur with significant inconstancy compared to the regularity of joint involvement in tabes and gliosis. And it is to be observed that they are not associated so definitely with extreme sensory losses as are tabes and gliosis.

The false arthropathies, as they may be called, are of quite different type from the genuine—they are distinctly inflammatory in nature, accompanied constantly by pain, redness, and often by suppuration.

In regard to those in hemiplegia, Weir Mitchell [12] has recently renewed attention to the intense irritative—not paralytic—sensory symptoms, and deprecates the super-

ficial reasoning which assumes a mere coincidence with rheumatism. These hemiplegic joint lesions are in most remarkable manner preceded, sometimes for years, by lively pains which are limited to the side afterwards paralyzed.

There are several other joint affections which may be spoken of as neuropathic. Among them the most interesting is *hydrops articulorum intermittens*. About thirty-five examples have been reported; they represent several different pathological states, but after eliminating those cases in which some such obtrusive condition as acute osteomyelitis gives at least an anatomical basis for the disease, if it does not afford an explanation of the periodicity, there remains a group in which the regularly or irregularly recurring hydrops, of apparently spontaneous beginning, is susceptible of no other explanation than that of vasomotor disturbance. Some of these are certainly hysterical. The location is far more commonly in the knee than elsewhere, but the other joints have been affected. It is usually unilateral, sometimes alternating right and left, seldom very painful, and often quite irregular in its manifestations. The more striking cases are however almost uniform in recurrence, and in duration of attack and in location. The intervals vary from hours to months, but most frequently last from ten days to two weeks. The disease sometimes persists for many years, finally to cease spontaneously or seemingly through the influence of treatment. This trouble is rarely mentioned in the books. Among others, Senator[13] has recently written upon it and points out that the question of the presence of slight degrees of real inflammation cannot be decided as long as we do not know the absolute distinction between exudate and transudate. An exceptionally well-marked case which I have lately seen will be made the subject of further report.

Another type of great interest is the acro-arthritis of Hutchinson, recently described further by MacMahon,[14]

regarded by the former as induced by gout in cases showing marked tendency to Raynaud's disease; that is, to conspicuous circulatory disturbance in the fingers. In this trouble the terminal finger articulations become typically wobble-jointed, with effusion, in tabetic fashion. The anatomico-pathological appearances correspond to those in tabes, which, it must be remembered, may itself produce arthropathies in this location. Tabes however is not present in Hutchinson's group.

The disease described by Marie as osteo-arthropathy of pulmonic origin is not an arthropathy to any essential extent, and is better described as ossifying osteo-periostitis with hypertrophy of neighboring soft parts. It is usually of toxic origin, and occurs in many other than lung diseases; although in those it develops the most acutely and to the highest degree.

Its essential lesion occurs in syringomyelia at times. Clinically, it begins with clubbing of the fingers, but the ordinary form of finger characteristic of lung disease is accentuated into a marked drumstick type with enormous nails, which attain the full width of the phalanx, leaving no marginal flesh. Decided or even cauliflowerlike tufting of the bone shows constantly, in radiographs. When fully developed, this disease induces pronounced enlargement of wrists, ankles and adjacent parts of the long bones. It is uncommon. The photographs which I show you indicate the freedom of the joints in the affection.

In regard to the treatment of the trophic joint lesions, unfortunately only too little can be said. But it is certainly true that it has been unduly neglected. The outlook when the affection occurs in tabes or gliosis is undeniably bad; but can we not do a little more in prophylaxis? In all severe nervous troubles, particularly when extensive paralyses of sensation accompany them, every precaution should be taken to prevent violence to the joints. There

should be constant watchfulness for grating or undue mobility; and with the first definite indication of approaching trouble in a joint, this watchfulness must be redoubled, and temporary rest in bed enjoined, with measures for immobilization. Relief of pressure from effusion must be secured in the event of tense swelling, by tapping. If done early in hemorrhagic cases the bleeding might be stopped by hemostatics, and the weakened joint structures saved from the destructive effects of great distension. Antispecific drugs should be given their slim chance in the beginning. The deleterious influences of ataxia should be guarded against by the early employment of Frænkel's invaluable method of training.

The extreme forms of joint disorganization with dislocations must have orthopœdic treatment; leather splints, properly applied, are very efficacious. Amputation is sometimes advisable, and I see in the peculiar state of trophic function no contra-indication. As a matter of fact, these joints and limbs heal readily. Indeed, it is often affirmed that the union of fractured bones takes place with unusual speed, the large callus being held to corroborate this view. This is wrong. The repair of bone occupies generally as much time as usual, indeed sometimes obstinate non-union is seen. The great callus, often observed, is the result of continued attrition in the ataxic, analgesic member.

The inflammatory type of arthropathy is more amenable to treatment. Suppuration demands surgical measures. As has been pointed out, there is not here the menace to life associated with other forms of acute suppurative arthritis.

For intermittent hydrops, besides removal of obvious cause of inflammation, the use of arsenic in long continued moderate doses has seemed to be of much service. But in the purely idiopathic variety, our present crude methods achieve little result.

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ARTICLE XXXI.

# TO WHAT EXTENT DOES "RHEUMATIC AND GOUTY DIATHESIS" ENTER INTO TRAUMATIC JOINTS (SPRAINS AND BRUISES), SEPTIC AND GONORRHŒAL JOINTS, ACUTE ARTICULAR RHEUMATISM, NEUROPATHIC JOINTS, ARTHRITIS DEFORMANS (OSTEOID, RHEUMATOID), AS AN ETIOLOGICAL FACTOR? WHAT IS THE SCIENTIFIC BASIS FOR SUCH A TERM?

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## TO WHAT EXTENT DOES "RHEUMATIC AND GOUTY DIATHESIS" ENTER INTO TRAUMATIC JOINTS (SPRAINS AND BRUISES), SEPTIC AND GONORRHŒAL JOINTS, ACUTE ARTICULAR RHEUMATISM, NEUROPATHIC JOINTS, ARTHRITIS DEFORMANS (OSTEOID, RHEUMATOID), AS AN ETIOLOGICAL FACTOR? WHAT IS THE SCIENTIFIC BASIS FOR SUCH A TERM?

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Of the six sub-divisions propounded for discussion under the common subject, "Diseases of the Joints often diagnosed as Rheumatic or complicated with Rheumatism," the one given above was assigned to me by your committee.

The question is composed of two distinct parts, the last of which calls for elucidation before the first can be logically considered.

Taking up first the question as to the scientific basis of the two terms "Rheumatic" and "Gouty Diathesis," before we can decide their true position in connection with scientific medicine, we are at once forced to analyze the origin and literal meaning of these terms in the light of our present chemico-physiological and pathological knowledge.

Take, for instance, the term "diathesis." What is its true significance in relation to the chemistry of the human economy? For years the term "diathesis" has been used to indicate that something has been handed down from generation to generation, but what that something is has never been made clear to the science of medicine. Taking the literal meaning of the word, from its Greek origin diathesis, and applying it to the chemistry of the body, it must

indicate simply and only that there is a tendency on the part of the animal economy to arrange the chemical phenomena of the system in certain and definite directions. This, however, does not justify the supposition that the individual is of necessity born to finally develop those symptoms recognized as constituting the conditions so long known under the term "gout" and "rheumatism." Therefore, the term "diathesis," as it is commonly applied, has neither fact nor foundation to rest upon; for all the evidence at our disposal tends to prove that all diseased conditions, with the possible exception of syphilis, are acquired after birth. All are abnormal chemical states of the system which can be either fed into or fed out of the human race, so to speak. This, however, is assuming a very high and almost theoretically ideal position: nevertheless, it is one that can be practically accomplished, provided we can succeed in attaining the highest perfection in the hygienic and dietetic conditions influencing animal life.

Thus we find, without going further into exhaustive details, that there is no real scientific basis for the term "diathesis" as it has been commonly used in the past. In fact, it would be better if this term were dropped entirely from medical literature.

In like manner the terms "gout" and "rheumatism," when viewed from a purely scientific basis, are almost as meaningless as is diathesis. Naturally, with this statement before us, the first question that arises is, what is this so-called "gout" and "rheumatism" that we hear and talk so much about? Turning back to the derivation of the word "gout" we find that it comes from the Latin word gutta, meaning a drop. According to Dunglison's Medical Dictionary, "gout received its name from the French goutte, drop, because believed to be produced by a liquid which is distilled drop by drop on the diseased part. The name was first used about 1270." Hence, gout is the drop disease.

This, however, gives no insight into the etiological factors entering into the production of the pathological conditions so long honored by this name. Nor does it give any suggestion as to the disturbances of the chemistry of the body, which will ultimately bring about the pathological changes and symptoms so well recognized under the name "gout." We might possibly strain a point and say, in a similar manner to what has been said in the past, that the term "gout" was coined to indicate the dropping of the uric acid radical into the protoplasmic structures of the various tissues of the body, as the result of the final and vicarious oxidation of the proteid molecule in the tissues already made pathological by the prolonged disturbances in the chemical activities of the animal economy.

Vicarious oxidation being synonymous with the term distillation of the ancient writers, referred to in Dunglison's definition of the disease:—accepting this more modern interpretation, there might be some justification for considering the term gout as a word based upon our present scientific data. On the other hand, as this vicarious oxidation of the proteid molecule in the already pathological structure of the body is but a little factor in the great and complex chemical problem constituting the condition so long known by the name "gout," there is no logical reason for regarding the term as one resting upon a clearly elucidated and perfectly scientific basis. Furthermore, the condition known as gout, as commonly defined, is said to be one in which there is a production of uric acid in the blood; a condition, however, that never occurs, because chemistry forbids the separate presence of an active acid in a strongly alkaline solution without the formation of a salt of that acid; at least it cannot occur unless all chemical laws are suspended in connection with the blood. So far as my observation and researches have carried me, there is nothing to warrant the assumption that the well known laws of

chemistry have been suspended, so that uric acid exists as such in the blood and does not result in the formation of a salt. Furthermore, it can be asserted that uric acid does not exist in the blood, for the reason that the salts of uric acid have never been found there, as must have been the case had uric acid entered the blood stream.

The definition above mentioned further affirms that the phenomena recognized under the term "gout" are due to the non-elimination of the uric acid from the blood stream, and its subsequent discharge from the blood as uric acid into the protoplasm of the various structures of the body, by preference into the cartilage cells of certain joints. But this explanation does not tell why the acid refuses to attack the sodium and calcium compounds in the blood stream, but seizes upon them with intense activity as soon as it is discharged into the semi-solid protoplasmic structures. Thus we find that the whole theory of gout is developed largely without any reliable scientific foundation, at least so far as the name is concerned, and it also disregards, almost from begining to end, the well known and inflexible laws of chemistry, as we understand them to-day. The only indisputable fact in connection with the whole theory of "gout," as it is commonly given, is the final deposition of the insoluble salts of calcium, together with some urate of soda crystals, in the various structures of the body. Therefore, it can very justly be stated that there is no well grounded scientific basis for the term "gouty diathesis."

When we come to the term "rheumatism" or "rheumatic diathesis," the mystery, so far as the derivation of the name is concerned, is greater even than is the case with gout. There is absolutely no scientific basis for the term. Yet under the term "rheumatism" is included a very great complexity of disturbed physiological phenomena.

If we attempt to secure any information through the derivation of the word we are led still further away from any

scientific basis for it. Taking its Greek origin, rheumatismos from rheum or rheo, and translating it literally "rheumatism," would mean a fluxion or flow, but of what or in what direction we are in total ignorance.

The more modern definitions are almost as vague and uncertain as those based upon the old and long since discarded humoral theory of rheumatism.

If there is no scientific basis for these terms, it is difficult to see how their relation to the topics under discussion can be elucidated or explained from a scientific standpoint. On the other hand, the natural inference is that both "rheumatism" and "gout" are, in a measure at least, connected with some of these joint affections under discussion.

Before we can discuss any etiological relationship between "rheumatic" and "gouty" conditions and the joint affections enumerated, we must first have some fixed and definite conception of the chemico-pathological phenomena that are to be included under these terms. The weight of evidence, however, both chemico-physiological and clinical, points to defective oxidation on the part of the system as the chief predisposing factor in bringing about these two dissimilar conditions.

In the majority of instances the chief cause for a sub-oxidation state of the system is, the ingestion of more oxidizable food than there is oxygen absorbed through the lungs to completely reduce the proteid constituents to the normal end products. In other instances, diminution in the food supply, together with that of a poor quality, may so deteriorate the nutritive activity of the system, that a pronounced anemia is produced, thus cutting down the intaking capacity of the animal economy for oxygen to such an extent that there is not sufficient oxygen absorbed through the lungs to perfectly oxidize the little proteid that is taken in the limited supply of food. Hence, too little or too much food may act as determining factors in producing an im-

perfect oxidation of the proteid constituents. It matters little whether the proteid be derived from the vegetable or the animal kingdom. Its imperfect oxidation and the toxic products, developed in consequence of incomplete oxidation transmitation, are *the* factors which excite the symptoms.

Attributing these pathological conditions to a suboxidation state of the system does not fully explain why in the one instance the result is "rheumatism" and in the other "gout." This was at one time attempted by assuming from the general clinical history that those individuals who partook freely of starches and saccharine substances developed "rheumatism," while those who lived largely upon an animal class of foods and used alcoholic beverages freely as a rule developed the condition called "gout." This was during the period in which lactic acid was supposed to be developed from the incomplete oxidation reduction of the saccharine elements and when uric acid was supposed to be the product of an animal diet only. That both uric acid and lactic acid do result from the imperfect utilization of the proteid constituents derived from the vegetable as well as from the animal kingdom, is a well established fact. With this conception of the oxidation problem it is easy to understand how both "rheumatism" and "gout" can be developed from an excessive diet of either vegetable or animal food. It does not explain fully, however, why a liberal vegetable diet is more likely to produce the condition called "rheumatism," and the liberal animal diet is more likely to produce the condition called "gout." At the same time it must be clear to every one who has observed many cases clinically that certain kinds of diet have a decided influence in determining the nature of the suboxidation processes. It should further be remembered that none of these incomplete end products found in the excreta is to be considered as constituting the disease any more than urea constituents normal nutrition. They should be regarded only in the light of

results or indicators by which the different types of suboxidation can be recognized and differentiated.

The development of the so-called rheumatic condition cannot be explained as it was at that time, by assuming that lactic acid is the chief etiological factor in producing the disease, and that this so-called lactic acid or rheumatic condition is due to the imperfect oxidation of the starches and sugar alone, while the gouty condition is due to imperfect oxidation of the proteid constituents. Assuming that such a condition is true from a chemical standpoint, it does not explain the development of the rheumatic condition, so-called, in those who live almost exclusively upon an animal diet, in which there is no excess of the starch and sugar to undergo imperfect oxidation reduction. Therefore, such a theory is absolutely untenable both from the chemico-physiological and clinical data at our command. Hence, we are forced to the assumption that in the so-called lactic or rheumatic condition, as in the so-called uric acid or gouty state of the system, all the toxic products found within the system, and all the abnormal products found in the excreta, are due to the imperfect oxidation reduction, or faulty isomeric transformation of the proteid constituents contained in the animal economy. This fact accepted, our theories can easily be made to fit all the clinical facts met with. Now, we can readily understand how it is that over-indulgence in the starches and sugars is so prone to excite imperfect oxidation of the proteid constituents. The starches, sugars and fats, all being easily and quickly oxidized into their complete end products, if taken in inordinate quantities will over-tax the oxygenating capacity of the system and there will not be left sufficient oxygen to complete the more difficult task of perfectly oxidizing the proteid constituents. In this manner they act as very potent factors in bringing about the imperfect oxidation of the proteid elements. Further than this, their oxidation being differently affected

than the proteid, it is easy to understand how their excessive utilization may have a different result upon the system than an excessive use of the proteid constituents; thus, in a measure, explaining why, in the one instance, we are more likely to have "rheumatism," and in the other the "gouty" condition.

As there seems to be so much confusion in the minds of many in regard to the true position and meaning of these imperfect products of proteid oxidation, and especially so in reference to the most common of all, namely uric acid, it may not be unprofitable to enter a little more into the detail of the formation of uric acid and its significance in connection with health and diseased conditions.

Uric acid is one of the substances by which nitrogen is eliminated normally from the system. It is an almost insoluble and very stable substance. It was first separated from human urine by Scheele, in 1776. It is a dibasic acid, having two replaceable hydrogen atoms, one of which is easily substituted by sodium, forming an acid urate, or biurate, of sodium. Both of the replaceable hydrogen atoms can be substituted by sodium, but, so far as is known, only the acid salt is a product of the animal economy. This urate is relatively soluble. Uric acid is formed by the oxidation of the proteid molecule or its derivatives, and is a lower oxidation product than urea, in the same manner that urea is a lower oxidation product than ammonia and carbon dioxide and water, which are the final products of proteid oxidation, chemically speaking. Why urea is the final oxidation product in man instead of ammonia, carbon dioxide and water, has never been explained, further than to state that it is according to the original plan of nature.

It has been claimed by some that uric acid is not an oxidation product, and they base their claim on the fact that this acid and its salts are the normal and complete excrementitious products in birds, reptiles, etc., in which animals

the oxidation powers are said to be exceedingly high. However, there is nothing to prove the capacity of birds, reptiles, etc., to take in oxygen, or of the blood to distribute this oxygen, and of their system to utilize it, and that it is sufficient to oxidize all the assimilated hydro-carbons and proteid compounds into their final and complete products. At any rate, to draw such an inference is begging the question. The chemical fact remains that their excreta, as compared with those of the higher order of animals, are products of a lower proteid oxidation; the same as urea is lower than the final product ammonia. Why in the one species the chemico-physiological apparatus of the animal economy is so constructed that uric acid and its urates are the final excretory product, while in another it is urea, is just as mysterious and absolutely unfathomable as the phenomena of life and death. In what follows we shall endeavor to show that in man the production of uric acid in quantities above the normal is due to a condition of sub-oxidation.

The two most prominent theories advanced to explain the genesis of uric acid in the system are:

1st. That it is an oxidation product manufactured in the renal cells.

2d. That it is made in some manner in the liver, spleen, etc., but chiefly in the liver.

Minkowski,[1] Schroder,[2] Horbaczewski,[3] are among the more recent defenders of the latter theory, while Garrod[4] is generally credited with the former. The latter view is the one that has been accepted generally and on the following grounds: First, that uric acid is found in the blood; second, that in gout it is found in the tissues; third, that after extirpation of the kidneys uric acid continues to be formed; and fourth, that at the height of digestion, when the liver and spleen are most active, uric acid is most abundant in the urine.

In this connection it may be remarked that in all the experiments which have been brought forward to sustain the theory that uric acid is formed outside the kidneys, or in the liver, there have been produced profound abnormal or pathological states of the system. Therefore they should be completely discarded from a physiological standard, as all deductions founded thereon are upon a purely pathological basis, and not normal.

The claim that is so commonly made that free uric acid is found in the blood is not tenable, as we have already seen; for, on coming into close relation with the alkali metals, salts of the same are immediately formed. Therefore, if this nitrogenous organic acid were in the blood, it would be in the form of a urate only, and as already stated such a salt has never been found in the blood.

The statement that uric acid does exist in the blood is based upon Haycraft's[5] method for detecting uric acid, or upon others of a similar nature. As they all admit of a possibility of oxidizing the proteid elements, and thus producing uric acid in the process for detecting this compound, they cannot be relied upon as proving the existence of uric acid in the blood. Some have claimed to obtain good results from this method. On the other hand, Salkowski[6] regards the process as of little value even in solutions known to contain uric acid, as the composition of the silver urate formed is not constant; this opinion is further supported by Gossage.[7]

On the other hand, it has been, and is still, distinctly affirmed that free uric acid and its biurate salts do exist in the blood; and the method called upon to explain the possible presence, in some form, of uric acid in the blood, is by the so-called "quadurate" (quadriurate) theory. The following statement made by Roberts,[8] the author of this theory, in his description of his method for obtaining the quadriurate, tends to prove that the quadriurate—if it really

has a chemical existence—is an accident rather than a constant product, for he says: "To obtain a product of uniform composition by this process requires a somewhat nice adjustment of the reaction. If the quantity of alkaline carbonate added be faint, the precipitate, on cooling, is apt to be contaminated with free uric acid. On the other hand, if the alkaline carbonate be added too freely, and the resulting alkalescence be excessive, the precipitate is apt to be contaminated with biurate. These risks are greatly minimized by using the alkaline acetates instead of the alkaline carbonates." From this it appears that Roberts failed to obtain the quadriurate with the regular uniformity necessary to establish his theory; in fact, the nice adjustment of the reagents required for the precipitation of the quadriurate might lead one to believe that, with this test, the precipitate was a mixture of biurate and uric acid in quantities to satisfy the theoretic demands for the quadriurate.

In Watt's[9] Dictionary of Chemistry (Vol. IV, p. 835, edition 1894) is the following: "Naha. ½ aq. (dried at 100) crystaline powder. Occurs as an amorphous uninary deposit." This statement, together with the fact that the alleged quadriurate is an amorphous deposit and very unstable, makes it appear highly probable that the biurate is still the form of urate most commonly found in the urine. It is not necessary to find a more soluble form of urate than the acid urate, for it is well known that the disodic monohydrogen phosphate,[10] which is often present in the urine in abundance, will hold large quantities of uric acid in solution. It is also known that the uric acid in the urine and in the absence any newly added chemical agent, steadily attacks the disodic monohodrogen phosphate, so that when the acid phosphate which will not hold the uric acid in solution has replaced the neutral phosphate, the uric acid is precipitated; all of which occurs naturally, and without adding anything to the urine as is required for the demon-

stration of the presence of the quadriurate according to Roberts's theory. The one occurs naturally, and the other requires very nicely adjusted reagents for its demonstration. This method, which may be called the natural one of precipitating uric acid in urine which apparently contains no excess of the acid, can be artificially quickened by a very simple procedure. This is best done by filling a test tube with urine and bringing the upper stratum to the boiling point, then add a drop or two of a four per cent. solution of acetic acid and set the tube aside in a cool place. At the end of a few hours, if there is an abnormal amount of uric acid contained in the sample and held in solution, as already described, the heat and the acetic acid will excite chemical action between the uric acid and the disodic monohydrogen phosphate, thus destroying the solvent power of the urine for the uric acid. Now the uric acid will rapidly be crystalized out, and the amount so formed, when collected on a filter and weighed, will give the exact percentage of uric acid in excess of the normal amount. In this manner and in a few hours the exact quantity of uric acid can be estimated. When the amount of uric acid is normal in the urine, no precipitation of the acid can be affected by this method. It then requires the use of a considerable quantity of one of the stronger mineral acids, sufficient to decompose the normal urates, and thus set free the uric acid otherwise held in combination with the sodium, before its presence is demonstrated.

The quadriurate, as has been said, is a very unstable compound and one that is easily broken up by washing with water. This being the case, its decomposition and the subsequent demonstration of the uric acid and the biurate in the blood should be easily accomplished. No mention is made, however, of finding the so-called quadriurate in the blood, but it is introduced artificially into the blood serum and synovia; and from this it has been claimed that

it can exist in the blood; but failure to demonstrate its presence directly in the normal blood stream is negative proof that it does not exist in that medium. The production of the quadriurate in the system necessitates first the production of uric acid. So far, in connection with the quadriurate theory, we are in total ignorance as to the point where the uric acid is manufactured, also as to where it attacks the sodium compounds to form the theoretical salt, and where the latter finally gains access to the blood. Until these doubtful points have been cleared up satisfactorily, the quadriurate theory is one grand speculative problem which does not explain the facts as developed by clinical observation. Unless our theories can be made of practical value in the study and management of the case at the bedside, they are of no real value.

From all the preceding, it can be justly deduced that uric acid and its urates have not yet been clearly demonstrated in the normal blood stream. That uric acid and its urates are found in the tissues in pathological conditions is true. This, however, does not necessitate the assumption that uric acid or its urates existed in the blood stream in defiance of all the known laws of chemistry. But it does demand the hypothesis that, as a result of a faulty nutrition, there is developed an imperfect oxidation, or an abnormal isomeric transmutation of the proteid compounds; that, as a result, these substances are oxidized at an abnormal position, instead of in the renal cells as normally occurs; that when this is the case there is a sudden production of uric acid and the formation of urates in the protoplasmic masses, and that when this happens all the symptoms are those which indicate a profound irritation and an increased chemical activity. Thus, by assuming a vicarious production of uric acid, a theory which holds true in many other abnormal and pathological conditions in the body, the presence of uric acid, or rather the urates, in the tissues is ex-

plained by simple chemical phenomena, and without deviating from any well-defined law of chemistry. All this can be done without predicating that uric acid or its urates are present in the blood.

The pathological condition which is produced by extirpation of the kidneys will admit of the formation of uric acid by the vicarious oxidation of the proteid substances in the protoplasm of cells other than those of the renal gland, upon the same principle as given in the foregoing example.

Upon the theory that uric acid is due to a deficiency in the quantity of oxygen reaching the body as a whole, and the renal cells in particular, the manufacture of uric acid by the kidneys should normally be greatest during digestion and least during the interval; for, during digestion, the carbohydrates, fats, and proteids are being introduced into the circulation in large quantities. The two former are rapidly oxidized, the one in the liver and the other in the lungs, tending to use up the oxygen supply, so that a deficient amount of oxygen reaches the kidneys and a less perfect oxidation transmutation of the proteid in the renal cells is the result, while the output of uric acid is augmented. Thus a normal physiological phenomenon, which is called upon to explain the production of uric acid in the liver instead of the renal glands, when viewed in this light makes it appear quite plain that no such deduction is justified. It rather strengthens the theory which points to the renal cells as the true source of the uric acid production, except in pathological states of the system, when it can be made at abnormal points by the vicarious action of the protoplasmic masses.

This theory of uric acid production by the oxidation of the proteid substances in the protoplasm of the renal cells is by far more logical, is best sustained by all the points in evidence, and fits most accurately all the known facts and conditions. This formation of uric acid is one method for the elimination of nitrogen from the system.

It is absolutely necessary to have this acid produced at this particular point, and poured into the uriniferous tubules; for by its action upon the disodic mono-hydrogen phosphate in the uriniferous tubules the mono-sodic di-hydrogen phosphate is produced, and the production of this acid phosphate of sodium is absolutely necessary to hold in solution the otherwise insoluble phosphate of calcium, thus preventing the formation of this form of calculi in the urinary passages.

The theory that the uric acid is produced in the renal cells in the manner described above is further substantiated by the steady presence of this acid or the urates in urine, and by their absence from the blood.

When uric acid is herein contrasted with urea as a suboxidation product, it is not meant to intimate that uric acid is the direct antecedent of urea. On the contrary, it is one of the complete end products of proteid oxidation, so far as the animal economy concerned, just as much as urea, carbon dioxide and water are final products. Chemically, less oxygen has been utilized to produce this particular form of compound than is required to produce urea and the other higher oxidation products. Physiologically, when produced in excess, it is a suboxidation product, for it is found that the output of uric acid is always augmented by anything that interferes with the utilization of the full amount of oxygen by the system, as compared with the quantity of food stuffs absorbed. A prolonged high tension of the arterial system under the influence of digitalis, for instance, causes the blood to be driven through the capillary blood vessels so rapidly that sufficient oxygen can not be taken up from the blood and utilized for the complete transmutation of the proteid elments of the food. As a result, suboxidation occurs, and the manufacture and output of uric acid by the renal cells are increased. The same is true with every condition of the system which produces

a prolonged high tension of the vascular system and a rapid pulse rate. When the intaking capacity of the lungs for oxygen is diminished by mechanical defects in the heart or by pneumonia, pleurisy, emphysema, etc., the oxidation of the proteid substances falls to an abnormally low degree, the excretion of urea decreases, and that of uric acid increases, the latter often to a marked degree. When the carbohydrates or fats are taken in excessive quantities, and especially the former, the oxygenating capacity of the system is exceeded, the more difficult task of oxidizing the proteid elements is imperfectly performed, suboxidation ensues, and as a result, uric acid increases in the urine, while the urea output decreases. When an excessive amount of the proteid substance is taken either alone or in combination with the carbohydrates and fats, the oxygenating capacity of the system is exceeded in a similar manner, and the proteids are imperfectly oxidized as before.

When the uric acid rises above the normal, it is simply a symptom found in the urine, which indicates an imperfect state of proteid oxidation and a general condition of malnutrition. This suboxidation may be, in a measure, caused by many different conditions acting singly or in combination—causes which may arise either in the nervous, digestive, circulatory, or in the respiratory system. In every instance there is more or less profound impairment in the glandular activity and nutritive tone throughout the whole system. In some instances one organ or tissue will suffer more than another, and thus the symptomic manifestations are varied and irregular in their development. When the renal cells temporarily cease to act, thus failing to produce uric acid, the associated defective state of nutrition results in many instances in the various masses of protoplasm throughout the body, which are in an abnormal physiological state, assuming an abnormal or vicarious action. In this manner the proteid substances are oxidized into uric acid

in the cells of the tissues with the immediate production of the urate of sodium. This abnormal oxidation may occur in any protoplasmic mass which is in this state of malnutrition, but it is most commonly met with in the cartilage cells of the metatarso-phalangeal articulation of the great toe. It does, however, occur in other parts of the body and in other tissues than the cartilaginous structures. This development of uric acid in the cartilage cells, or at other points of the body, results in its immediate precipitation at such points as a urate of sodium. This abnormal deposit acts as an intense chemical irritant, and excites a local inflammatory process by which a fibroplastic exudate is thrown around this foreign body until the urates are finally incapsulated, and gradually all the local symptoms subside.

The problem has further been very much simplified by the fact, now generally recognized, that all the non-nitrogenous food products, such as starch, sugar and fats, are directly oxidized into their end products, carbon dioxide and water, and that they do not yield any toxic by-products to the system. Therefore, we are left to deal chiefly with proteid constituents as the etiological factors in the production of these toxic and suboxidation conditions of the system. This much established, we still have no clear and complete explanation for the different degrees and forms of suboxidation processes which constitute the various diseases, such as so-called "rheumatism," so-called "gout," etc.

Strenuous attempts have been made in this bacteriological age to associate a specific germ with all pathological problems, and in many instances this has apparently been sucfully accomplished. Still, the declaration of the presence of a specific germ as the causative factor in the production of disease does not explain in full the special and characteristic features of the pathological lesions found, or the symp-

toms by which the different diseases are characterized. Working along these lines, a considerable number of observers profess to have found a specific germ as the causative factor in the production of so-called "rheumatic conditions" of the system. Among this number may be mentioned Von Schueller,[11] Buss,[12] Brunner,[13] Buday,[14] Petrone,[15] Hlava,[16] Tizzomi,[17] Goldscheider,[18] Lœffler,[19] Boulloche,[20] Mantle,[21] Guttmann,[22] Fleischhauer,[23] Waibel,[24] Bouchard,[25] Sahli,[26] Lucatells,[27] Leyden,[28] Singer,[29] Chvostek,[30] and Humphrey.[31] All of these observers have given more or less attention to this subject. Buss states that he considers it highly probable that acute articular rheumatism, so-called, is in many, perhaps in the majority, of cases caused by attenuated pyogenic microorganisms, among which he includes the Friedlaender pneumobacillus and the diplococcus of Fraenkel-Weichselbaum. Sahli cultivated streptococci from the blood and contents of affected joints in cases of so-called acute articular rheumatism. Singer made bacteriological examinations in seventeen cases of so-called articular rheumatism and secured positive results in sixteen instances. In ten cases he found staphylococcus albus; in one, staphylococcus aureus; in three, streptococci; in two, staphylococcus albus and streptococci. He further argued from his observations that the constancy of the occurrence of the microorganisms in the urine, and the fact that the number of the colonies obtained, diminished in number as the symptoms improved, were sufficient evidence for considering a coccus infection as the exciting cause of the disease. Chvostek, on the other hand, made repeated observations, in twelve cases of so-called rheumatic affections of the joints. In one of these cases he found the diplococcus urea; in another, in which the urine was not drawn with a catheter, staphylococcus albus; and in a third, large cocci, which have not yet been described, but which, probably, according to Chvostek, are

derived from the urethra. In this connection it should be remembered that Krause[32] has shown that bacteria are often excreted in the urine during infectious disease, which have no connection whatever with the particular infection from which the patient is suffering, and which must not therefore be regarded as the exciting cause of the disease. Among these bacteria the staphylococcus albus plays an important part. And this is the form of microorganism that has most frequently been described in connection with these rheumatic conditions. Krause further says that these bacteria are no longer found in the urine when all the signs of disease have vanished, simply, as he argues, because the conditions of the system necessary for their growth have been removed.

Chvostek extended his examinations and made examinations of the blood, urine and articular fluid, in cases of so-called acute and chronic articular rheumatism, as well as in cases of other infectious diseases, in which an acute swelling of the joint takes place. The results which he obtained from his examination of the articular fluids were negative in all his cases, except in the changes in the joints that arose directly in the course of a sepsis or a gonorrhœa. Chvostek sought further to discover whether the bacteria which are present in the blood entered the joints during the life of the patient; and if so, the conditions necessary for their transit through the wall of the blood vessels. In this connection it may be mentioned that his experiments on animals showed that certain alterations in the nutritive composition of the vascular wall must be produced before the passage of the microorganism could be effected. He also demonstrated, to his own satisfaction, that the construction of the synovial membranes and that of their contained blood vessels resisted to a high degree this nutritive change that makes possible the passage of the germs, thus rendering it very difficult for the microorganisms to pass through

the walls of the blood vessels and gain access to the joints. This is not the case, however, with the vascular walls of the kidneys; hence, the bacteria, if they appear at all in the joints, will do so at a much later period than they are excreted by the kidneys.

That microorganisms have been found in the urine, and in some instances in the blood, and in the fluid accumulations in the joints, and in various structures of the body, in connection with the varying conditions which have been described under the common term rheumatism, cannot be denied. We may even go further and state that the prevailing organism when described at all is most frequently of the coccus type.

From the great diversity of results obtained, and from the fact that no one form of microorganism is found with any degree of regularity in connection with these so-called "rheumatic" conditions, the consensus of opinion is that the pathological conditions and symptoms classed as rheumatic are not to be attributed to the direct and intrinsic bacterial invasion of the structures of the body, as is the case in connection with some of the well known and undisputed microbic disease. Nevertheless, it is reasonable to suppose that in conjunction with over-feeding, and with under-feeding, and the consequent suboxidation state of the system, the action of the microorganism and the toxic products that are developed as a direct result of their presence in the alimentary canal, are largely responsible for the different forms and degrees of suboxidation included under the terms "gout," "rheumatism," etc., etc. The microorganism in all these instances acts by its presence in the alimentary canal, and by its disturbing influence upon the digestive process, and not by its inherent presence within the intrinsic structures of the system.

To understand this proposition thoroughly, it must be remembered that the presence of certain kinds of bacterial

life are, in all probability, absolutely essential to a perfect performance of the digestive function. In this connection it must also be remembered that only a few years ago Nuttall and Thierfelder,[33] in a series of experiments in which they used young guinea pigs, apparently proved that the alimentary canal could be kept free from all bacterial influences, and a perfect nutrition at the same time be maintained. In opposition to this is the fact that in almost all instances the alimentary tract at all times contains various microorganisms. Added to this are the more recent experiments of Dr. M. Schottelius.[34] Dr. Schottelius' series of experiments was carried out in a manner similar to those of Nuttall and Thierfelder, but instead of selecting the guinea pig, hens and their eggs were utilized for the experimental work. Two sets of eggs were taken, one in which the surface of the eggs was made absolutely free from bacteria by washing with a strong solution of bichlorid of mercury, the remaining set remaining contaminated by microorganisms. The first, so far as their exterior is concerned, will be called for convenience sterilized eggs, while the other batch will be designated as nonsterile eggs. The former were introduced into a sterile incubator, while the others were not. When the chickens were hatched, those from the sterilized incubator were carefully fed apon sterilized food, while those of the control experiment were given ordinary food. The feces were carefully examined in both instances. In the first set, or the so-called sterile chickens, the feces were found to be absolutely free from all forms of bacterial life, while the reverse was found to be the case in the control experiment. At the end of a certain period of time the nonsterile batch of chickens were found to have gained in weight much more rapidly than the sterile batch. The gain was 250% greater in those in which the bacteria gained access to the alimentary tract. Further than this, all of the so-called sterile chickens died within three weeks

after they were hatched, while the control chickens lived on as usual. Thus it would seem to be clearly proved, that for the most perfect working of the digestive function in the alimentary tract, and also for assimilation, the presence of certain kinds and amounts of bacterial life and their chemical products in the alimentary canal are absolutely essential.

To determine which form of bacterial life is essential for the maintainance of a perfect physiologic state, will require a long series of experiments, in which the composition of the food is accurately determined before being ingested. At the same time the excreta, representing the utilization of the foodstuffs and tissue waste, must be accurately studied. Then the various kinds of microorganisms at work in the contents of the alimentary tract must be determined through a study of the bacterial life found in the feces. With this there must also be associated an isolation of the various kinds of bacteria found in the contents of the alimentary canal in connection with the condition recognized ordinarily as the normal state. Then the action of the bacteria should be carefully studied on the normal sterilized and nonsterilized foodstuffs outside the body; and this must be done with the bacteria, singly and in combination. Their action should also be studied upon the food in the presence of the various digestive ferments. When all this has been accomplished, a very fair knowledge of the action of germs in the digestive process in the normal state will be secured. Then, in a similar manner, their action upon the digestive process could be studied in connection with the conditions designated as intestinal indigestion, and also in connection with all the pathological processes which appear, in a large measure at least, to take their origin in an imperfect digestion and assimilation, and in which an incomplete oxidation is a leading feature of the disease, as occurs in the so-called "gout," "rheumatism," "Bright's disease," "diabetes," etc.

While all this desirable information is not as yet at our command, there is sufficient evidence at hand to warrant the assertion that the varying degrees and kinds of suboxidation met with, such for instance as the so-called gout and rheumatism and a host of other conditions, are the result of some special kind of microorganism acting in the alimentary canal; or they may be the result of two or more varieties acting together; or may it not be that the absence of certain kinds of bacterial life is the determining factor in the production of the diseased process? In either instance it it may be assumed that abnormal or toxic products are produced in the alimentary canal, which, when introduced into the circulation with the foodstuffs, act as the direct and determining factor in establishing the special degree or form of suboxidation that we know by these time honored names "gout," "rheumatism," etc. By assuming the development of a variety of toxic products it easily explains the great variety in the clinical picture as witnessed under the so-called rheumatic condition.

This line of argument is sustained, so far as the symptoms are concerned, by the fact that with the introduction of certain chemical compounds into the system definite symptoms follow, as when morphin, strychnin, atropin, pilocarpin, etc., are introduced. Why morphin, strychnin, atropin, pilocarpin, etc., are always followed by a pretty uniform train of symptoms depending upon the special one used, has never been absolutely explained. Yet no one attempts to deny the fact. In a similar manner it is reasonable to suppose that the symptoms of disease are the result of the introduction into or development within the system of definite chemical compounds; that these chemical products either emanate directly from the microorganism or are produced from the proteid constituents, in which the bacteria grow, by the action of the bacteria upon the proteid molecule. This latter view ap-

pears to be the more probable taking all things into consideration. The modification of the physiological phenomena by the introduction of the varying pathological conditions met with; just the same as the introduction of chemical compounds can be made to modify pathological phenomena, and thereby enable the system to be brought back into a more nearly physiological condition. In all these suboxidation diseases the action of the bacteria is on the proteid constituents as they exist in the chyme rather than upon those which exist as intrinsic parts of the body.

Thus we find that the two great predisposing factors in the development of so-called gout and rheumatism are the prolonged intake of a larger amount of nutritive pabulum than the system can perfectly oxidize or conditions that so reduce the oxygenating capacity of the animal economy that the small amount of food taken cannot be perfectly oxidized. Added to this, as the exciting and determining factors in the production of the special type encountered, are the action of the bacteria on the proteid in the alimentary canal and the formation and absorption into the system of toxic products in conjunction with the food products absorbed. These two factors acting together determine the form of the suboxidation, the nature of the pathological lesions, the character of the symptoms, and the abnormal and by products that are found in the excreta. It is largely by the latter that we are enabled to accurately differentiate between the different forms of these suboxidation conditions that afflict humanity. In conjunction with the two predisposing factors and the exciting factor in the production of these suboxidation conditions there are a few other things that must not be overlooked, as they often exert a decided influence in determining the final result. They are the varying changes in the temperature, hygienic surroundings, nervous disturbances, etc., for they are all very important factors and help to disturb the perfect working of the glan-

dular system. Just how the modus operandi of this bacterial factor working in the alimentary canal can best be absolutely determined is the question that is still pressing for an answer from the physiological chemists. Until it has been fully solved, the absolute etiology of these suboxidation processes under discussion, so-called gout and rheumatism, cannot be absolutely elucidated except in theory. But even in the absence of this much desired complete verification, this explanation, as here given, adheres more closely to all the facts thus far known to modern science than any explanation previously advanced.

With this conception of the chemical conditions that are at work with the system, when affected with so-called gout and rheumatism, the etiological relation of these conditions, if any exists, can be applied to the various diseases that are under discussion. It is, nevertheless, difficult to comprehend how false feeding, the action of the microorganism in the alimentary canal, and a toxic infection of the system therefrom, can in any manner be looked upon as a primary etiological factor in the production of a traumatism to a joint, a sprain, or a bruise. Therefore, it must be asserted in the very beginning that there is no direct primary etiological relationship between the so-called rheumatic condition of the sytsem and traumatic lesion of the joints. On the other hand, it is equally true that the abnormal chemical condition of the system which produces that state of the the animal economy classed as "rheumatism" and "gout," will assert itself, and modify the whole course of the pathological problem in connection with a traumatism. This does not warrant the supposition, however, that the so-called rheumatic state of the system in any manner acts as a primary etiological factor in producing the traumatism of the joint structures. Penetrating a little more deeply into this intricate problem, we find that the products absorbed from the alimentary canal, and which, if retained in the system,

are the determining factors in producing the special type of suboxidation that excites the lesions and symptoms classed as rheumatic, will influence the pathological changes following a traumatic injury to the joint. This is assuming, however, that there has been false habits of feeding, micro-organisms at work in the alimentary canal, absorption of toxic products into the system, and that, in consequence thereof, the animal economy has been or is on the verge of the suboxidation state called rheumatism, prior to the receipt of the injury. In other words, after a mechanical or macroscopic traumatism to the joint structures, the nutrition in the injured parts, in one who is suffering from this characteristic chemical state of suboxidation of the system, which just precedes and makes possible the so-called rheumatic condition, will undergo changes different from those occurring in a more normal state of the system. When this term toxic product is used in connection with the diseases under discussion, it is understood that one of two conditions exists; either the system contains one or more by products which have resulted from an incomplete oxidation reduction of the proteid elements, or the system contains proteid molecules which instead of pursuing their normal isomeric transformation course, have been deflected therefrom. When this latter occurs, the proteid molecule is so changed, isomerically, that it becomes toxic in its character, yet it still remains an unoxidized proteid body. This irregular isomeric transformation of the proteid bodies, and their change into a toxic form, easily explains the toxicity of the system. It further shows the difficulty, if not the absolute impossibility, of detecting the presence of these toxic proteid bodies as they circulate in the blood and lymph stream. In whatever manner produced, the toxic product which, prior to the injury, was eliminated from the system without exciting any active symptoms, will often aid in intensifying the pathological changes and

symptoms resulting directly from the traumatism. Lesions and symptoms which would not have developed in the absence of the traumatism, are produced by virtue of the presence of the suboxidation and toxic condition of the system called "rheumatic." On the other hand, they would not have occurred in the presence of the traumatism in a system perfectly free from so-called gouty and rheumatic conditions. In instances of this character, the so-called gouty and rheumatic states of the system can be looked upon as etiological factors in the production of some of the pathological changes and symptoms which follow direct traumatisms to the joint structures. They are, however, always secondary and never primary.

This relationship of the so-called gouty and rheumatic condition of the system should always be taken into consideration in connection with every traumatism that implicates the joints. If such a state of the system is found to be present, it must be given due consideration in the general management of the case; otherwise recovery will be greatly retarded.

In one instance that came under my observation, a severe wrench to one of the great toes had occurred in a gentleman in his early fifties. This injury was followed by all the characteristic symptoms common to an ordinary attack of so-called subacute or chronic gout. So long as the case was treated as one of simple traumatism, which was the method pursued before he came under my care, there was no pronounced or permanent improvement. From the moment the condition was regarded as being primarily traumatic in its origin, and, in a large measure, secondarily kept in motion by the gouty suboxidation condition of the system which preceded the traumatism, and the treatment modified in accordance with this latter view, improvement was steady, and a permanent cure was speedily effected. Here the traumatism precipitated the so-called gouty attack,

if I may so state it, and the only line of treatment that could be effectual was one directed toward removing the suboxidation state of the system. This is only one of many cases that could be cited in illustration of this principle. The same holds true in connection with the so-called rheumatic conditions. On the other hand, we frequently meet with instances in which slight fractures running into the joints and dislocations of the carpal and tarsal bones are diagnosticated as so-called rheumatic affections. Thus we find that there may be a commingling of the two conditions, both acting as etiological factors, even in connection with simple traumatic injuries of the joints. Therefore, each and every case examined must be carefully analyzed by itself, and due consideration given to the traumatism and to the possible influence that a previously existing suboxidation state of the system may at the time exert in the production of the lesions and symptoms. When this is systematically done, treatment will be more scientifically applied and the restoration of the involved joint to its normal functional activity more speedily effected.

With the septic and gonorrhœal affections of the joints, all that has been said as to the relation of so-called gouty and rheumatic conditions of the system to a traumatism can similarly be applied to these two diseases. The so-called gouty and rheumatic states of the system are in no sense primary etiological factors in the production of a septic or so-called gonorrhœal joint. Both, however, may play a secondary part in determining the nature of the lesions and the character of the symptoms as in the simple traumatic affections of the joints. But their influence will not be as well marked as it is in the former instance. This is especially true in connection with a septic joint, where the intensity of suppurative process and its symptoms mask all those of a minor character, and also because death usually occurs early in the disease. The same may be said in con-

nection with so-called gonorrhœal infections of the joints; for it is still an open question whether the acute joint symptoms developed during the course of the gonorrhœal, or following as sequelæ, are not due to a direct septic infection instead of a gonococcus invasion[35] of the joint. With so-called gonorrhœal rheumatism which develops many months after the acute gonorrhœal attack, the suboxidation state and the toxic involvement of the system through the alimentary canal are in all probability more largely responsible for the pathological changes and symptoms than is the gonorrhœal infection. Thus the so-called rheumatic condition may play a large part in the development of the lesions and symptoms in so-called gonorrhœal affections of the joints; but it is always a secondary and never a primary cause. Still, in the management of all these cases it must be carefully considered and the treatment directed accordingly if the best results are to be attained.

When we come to the consideration of those conditions commonly classed as acute articular rheumatism, we for the first time reach a point in which the errors in diet, the action of bacteria in the alimentary canal, and the absorption or the development of the toxic products directly traceable to their presence, act when they are introduced into the system as the direct etiological factors in determining the special type of suboxidation. In this manner we can further look upon the suboxidation and toxic invasion of the system from the alimentary canal as the direct and exciting cause of the so-called gouty and rheumatic affections. The great variety of toxic products which can be produced in this manner in the alimentary canal, and that can be developed within the system, as well as those absorbed from the alimentary tract, can be made to explain the varying degrees and types of suboxidation encountered. They can also be made to explain all the different forms and types of so-called rheumatic affections that are encountered clinically. This

theory of the production of these lesions is well supported by that of Weigert[36] in his explanation of the various kinds and types of renal lesions that are known to exist. As yet, however, we are not sufficiently familiar with the micro-organisms and the toxic products to fully trace the *modus operandi* from the point of origin of the etiological factor to the final result. If, as seems quite probable, many of the toxic products are simply isomeric forms of the normal proteid molecule, it will be, as before stated, a very difficult matter to trace, isolate and identify them. At the same time, from the data at our command, it seems reasonable to assume that continued errors in diet, the action of various kinds of microorganisms in the alimentary canal, and the production of an almost endless variety of these toxic products, can easily be made to explain the many forms and types of the so-called rheumatism, even from the slightest myalgia to the most extensive destruction of the joint structures. Even the so-called neuropathic joint affections can be elucidated upon this theory of a disturbance of the nutritive functions of the animal economy, as they can in no other way. When one kind of toxic product affects the system, it, like some of the well known drugs already mentioned, will influence the chemistry of the body in its special manner, and a certain definite train of lesions and symptoms will follow. The introduction of still other kinds will produce their special results, and an almost endless variety of examples might be mentioned. In the one instance the abnormal and toxic condition may result in a simple congestion only of the intermuscular plains, with an undue pressure upon the nerve endings distributed to that particular part. Then we have the condition called "muscular rheumatism." In another instance the abnormal and toxic condition, by virtue of a difference in the toxic product, will implicate the central or the peripheral nervous system and give rise to that class of cases which are known as

neurotic and in which there is no very pronounced joint lesions. In still others the toxic elements will cause a more profound disturbance of the peripheral nervous mechanism, and thereby the action of the so-called trophic centres will be so profoundly disturbed that actual and pronounced destructive changes in the joint structures are produced, such as are found in Charcot's disease. The more common lesions, however, that occur as the result of this special type of suboxidation and toxic infection of the system classed as rheumatism, is one in which the vascular areas in and around the joints become congested and are followed by a pronounced edematous swelling of the soft structures in and around the joints affected. In all these cases it is the lowered nutritive activities and the action of the toxic products within the system, acting either upon the central or the peripheral nervous system, that cause the disturbance in the vascular and nutritive supply to the parts affected and thus give rise to the anatomical changes and symptoms. In the vast majority of instances the injury to the structures involved in the so-called rheumatism is not sufficiently great to excite the phenomena characteristic of a true inflammatory process. Hence, with the removal of the cause and an improvement in the general, as well as in the local, nutrition, there is a perfect restoration of the implicated joints to their normal state. In a few instances the primary injury from the toxic infection may be sufficiently great to excite a truly inflammatory process with all the changes characteristic of inflammation. In all these cases there is a tendency to more or less permanent damage to the structures in and around the joints involved. The amount and permanency of the damage to the joint will depend entirely upon the intensity of the primary injury and the duration of the actual inflammatory process.

The most extensive damage to the joints, however, is met with in those cases which are not inflammatory, but in which

there is a long-continued general suboxidation state of the system together with its pecular type of toxic infection. In these instances the continual augmented vascular supply to one area, with a defective supply to another adjacent zone, but in both instances of material poor in quality and also toxic in its nature, causes a very irregular distribution of the nutritive pabulum with which to carry on the functions in and around the joints. So great and varied is this abnormal distribution of the nutritive pabulum, that almost all grades of new formations are met with in and around the joints. These vary from simple swellings consisting of atrophied fibrillated connective tissue infiltrated with a watery exudate into its meshes, to well defined enlargements composed of dense fibrillated connective tissue almost as firm and well developed as the normal tendon tissue. These new formations are located chiefly in the ligamentous structures and around the end of the bones at the point of the attachment of the ligaments and tendons.

This newly formed tissue in some cases is infiltrated with the insoluble salts of calcium and the urates, but it differs from the so-called gouty depositions in position rather than in kind. In the latter the salts are deposited by preference in the cells of the cartilage structures, while in the condition under consideration it is outside the joints and in the newly formed tissue.

In some rare instances there may be developed both a carious condition and the development of new bone in and around the joint. The two latter conditions are very rare and always suggest the possibility of a syphilitic infection as an accessory etiological factor.

In all but this latter class this complex chemical suboxidation problem, with its associated toxic condition of the system which has for so many years been included under the so-called rheumatic condition, can be looked upon as the direct etiological factor in producing the lesion and

symptoms of so-called acute articular rheumatism in all its varied forms, also the neuropathic joints and some of the forms of arthritis deformans. This is especially so in all cases in which the joints become enlarged and distorted by an undue and irregular deposition of newly formed connective tissue.

In the osteoid types and in those cases in which a certain amount of caries occurs, it is highly probable that other etiological factors must be taken into consideration in explaining the loss of bone and the development of new bone. As already mentioned, it is highly probable that syphilis plays a decided part in producing these grave changes, as it is well known that syphilis causes a decided tendency on the part of the system to bring about a carious state of the bones and a tendency to the formation of new bone.

This method of explaining the phenomena that occur in connection with conditions of the system so long known under the names "gout" and "rheumatism," places us in a much better position to prevent their development, and to displace them from the system when they have developed. It further shows in which of the diseases under discussion the "gouty" and "rheumatic" condition of the system acts as a direct etiological factor; and also where its influence is only of a secondary nature.

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## DISCUSSION.

Dr. Joel E. Goldthwait, of Boston:—I am personally grateful to Dr. Porter for the presentation of this subject. He puts the subject on a very different plane from the treatment which it receives in most of our text-books. I am particularly glad that he mentioned trauma as a factor of these joint conditions as carefully as he did, because it seems to me that is a fact which should be borne in mind in the management or in the treatment of comparatively slight injuries in persons at or past middle life. You all know how common it is for individuals to have rheumatism after they have had a sprain. It seems to me it is so common that in our prognosis and in our treatment we should recognize it, and my custom is to state this possibility to the individual and plan treatment with the idea of preventing the condition developing later which is so commonly seen. You know how common it is for a wrench or sprain to be well in two or three weeks, or in a month or two months, and then for the patient to be crippled; and the form which is most commonly seen in those conditions is the osteo-arthritic type.

In my own experience the rheumatoid type following trauma has been a very rare condition if seen at all.

One other addition to my contribution,—the fact that glandular enlargement is not seen in the osteo-arthritic type, but is seen in the rheumatoid type.

I was particularly sorry not to hear Dr. Jack's paper, but I would like to add one point in the discussion of acute articular rheumatism, which is simply this: that in a considerable number of cases after the acute inflammatory condition has subsided one joint may remain swollen, and in spite of compression, salicylates and all kinds of treatment the swollen joint does not improve. In several cases which I have seen, the joints have been opened, and in all of them the apparent swelling has been due to the presence in the joint of coagulated fibrin. For some reason the exudate has not been absorbed as in the other joints, but the fibrin in the exudation has been precipitated and has formed a clot, and this has represented a foreign body, which, slipping about, has kept up the irritation and you have a traumatic synovitis after an acute articular rheumatism. Upon the removal of the coagula all of the joint symptoms have been relieved and the patient has been well. This fact that the fibrin can be precipitated from the synovial fluid is a thing which should be remembered in any kind of joint inflammation as an explanation of why the swelling does not go down.

ARTICLE XXXII.

## THE ANNUAL DISCOURSE.

# MEDICAL PROSPECTS.

By GEORGE E. FRANCIS, M.D.
OF WORCESTER.

DELIVERED JUNE 12, 1901.

NOTE.—At an Adjourned Meeting of The Massachusetts Medical Society, held Oct. 3, 1860, it was

*Resolved*, "That The Massachusetts Medical Society hereby declares that it does not consider itself as having endorsed or censured the opinions in former published Annual Discourses, nor will it hold itself responsible for any opinions or sentiments advanced in any future similar discourses."

*Resolved*, "That the Committee on Publications be directed to print a statement to that effect at the commencement of each Annual Discourse which may hereafter be published."

# MEDICAL PROSPECTS.

Mr. President and Fellows
of The Massachusetts Medical Society:

As our Society gathers for its first meeting in the Twentieth Century, the thoughts of all of us are naturally turned into one channel. A year ago we were led to survey the wonderful achievements of the century just ending; to-day brings the far different task of attempting to foresee what the future has in store for the profession of medicine.

I have not been so presumptuous as to attempt to predict the *scientific* advances which medicine is about to make; my purpose to-day is simply to note some conditions and tendencies which are now to be observed in the *practical* side of medicine, and to forecast, if may be, some of the changes which they will bring about.

The most obvious, and perhaps the most important new factor in medical life, is, of course, Specialism. I speak of it as new, because many of us remember the time when practically it was not, and have almost witnessed its birth and its growth. It is doubtful if in the year 1861 there was a single member of our Society who limited

his practice to any specialty except that relating to the eye. In the Harvard Medical School of that date, every professor was to some extent a general practitioner; the one exception to this statement being the illustrious professor of anatomy. If Oliver Wendell Holmes had ever actively engaged in medical life we may be sure he would have shunned and scorned any appearance of putting a fence about the range of his studies or his work. In one of his latest books,* he makes a good deal of fun over some of the abuses of specialism; but were Dr. Holmes with us to-day, he would be among the first to give hearty and grateful recognition to the magnificent additions to our science which the workers in special lines have lately given, and still give, and very certainly will continue to give.

We have been speaking of specialism as an innovation, forgetting that it is but a revival from very ancient times. Baas, in his History of Medicine, tells us that in Egypt, "according to the account of Herodotus, there were physicians [who were all priests] for every part of the body. This specialism is *per se* an evidence of a civilization of high development, indeed of one tending toward its downfall, and in Egypt it attained a perfection which our own system, with all its completeness, has not yet reached. We know that the sick were visited and treated at their homes by the physicians. The latter must first however be sent for to the president of the temple, who selected and

* Over the Tea Cups.

despatched to the patient the specialist best suited to his case."

It seems to be true that specialism is absolutely essential to progress in every department of human thought and activity. As knowledge has broadened, it has become more and more impossible for a single brain to keep up in all, or in many, of the lines of advance, especially in science. To-day we are leaving scientific progress out of the discussion, and are considering how the practical side of medicine is likely to be affected by such a new condition.

When specialism is applied to manufacture, it is termed "division of labor." It seems to be demonstrated that the best results at lowest cost are only to be had when each artisan and operative has become trained in a narrow specialty, so that he can do his own little range of work with the utmost rapidity and accuracy. The making of shoes is one of our great industries, and many of our people earn their living thereby. Each shoe is the final result of successive bits of labor by scores of specialists. There seems to be no doubt that this is the cheapest way to make good shoes. But among all the skilled workmen who combine to make the finished product, there is not one who is taught to make a whole shoe by his own labor. There seems to be no place for the cordwainer of former days.

Is there perchance a useful parable here?

All specialism, whether mental or manual, seems to narrow the range of one's powers. It is sim-

ple prudence therefore to study this possible danger threatening our profession, and to do what we can to counteract the injurious effects which are likely to attend the spread and sway of specialism. We cannot expect, even if any have the desire, to check this spread and sway; the new way is necessary; it is inevitable; by it alone can scientific progress be made. And yet it bears with it some dark and threatening shadows.

Imagine, if you please, a visit made about this time to his Alma Mater Medica, by R. Van Winkle, M.D., Harvard, 1861. Picture to yourself his amazement at the growth and development of the school he once knew so well. What, do you think, would be his predominant feeling? Not wonder at the growth;—not admiration of the development;—not envy of the superior advantages of every kind;—though no doubt all these thoughts would arise in turn; his final sentiment, if I mistake not, would be of pity for the unfortunate student of to-day, whose tasks have become so much more extensive and difficult.

The purpose of a good medical school is to fit each graduate thoroughly and completely for the successful practice of medicine; and to accomplish this result two distinct processes have to be carried on at the same time. Each pupil must be *educated*, in the strict sense of the word; that is to say, all his faculties of body and mind must be developed and trained so that when he enters upon his life's work he can observe accurately, can compare things rightly, can reason and decide logi-

cally, and can dexterously carry out the details of treatment.

At the same time it is equally necessary that he be *informed;* that his memory be stored, crammed full, with facts of many kinds which it is essential that he should retain, ready for instant use, through his whole professional life. These medical facts are not self evident things; they must be learned laboriously, and laid away in the storehouse of the memory with care and system. Every school of applied or practical science is forced to keep in view this double object, of educating the faculties, and of storing the memory with selected facts for future use.

Now, all the changes and improvements which have been made in the medical schools;—the laboratory work, teaching in small sections, individual clinical training, and the rest;—all these conspire to bring about this result among others;—that from the hour of his matriculation until he receives the coveted parchment, the student is scarcely ever free from the close personal influence of *specialists.* All the instruction he receives which is fresh to him, not to be found in the text books, is from specialists. Every case which is any way striking or unusual, is sure to be referred to some specialist for diagnosis and treatment. In passing we may observe how strongly all this is likely to bias the youthful mind and to foster the early choice of a specialty as a career.

Medical training on these lines is admirable from many points of view. It provides a teaching staff not only thoroughly well informed but full of fresh enthusiasm and vitality. The pupil is inspired by the consciousness that he is in the very current of progress.

An oft-quoted maxim is to the effect that the ideal professional man should know all about one thing, and something about a great many things. This is really a paraphrase of what has just been said, that the student in practical science must be educated and also informed. There is probably no better way of getting the education than by good advanced work in one, but only one, special line. We can appreciate how difficult are the problems which the heads of medical schools have to solve, in this matter of limiting judiciously the amount and variety of advanced special work done by undergraduates, and especially in properly guiding the choice of facts which are to be memorized.

There comes to mind a plaything of childish days, which we used to call the "dissected puzzle." A map or picture was pasted upon a thin piece of wood which was then artfully cut up into a number of pieces of very irregular outline. We used to think it amusing to fit these parts together. In the medical schools we find the domain of medical knowledge divided up, more or less artificially, into many separate departments, which vary greatly in size and importance. The practical

problem or task is to combine these parts into a complete, harmonious and useful whole.

The comparison is far from perfect, as we appreciate in a moment, and yet it may serve the present purpose. The various branches of medical instruction are not bounded by clean cut lines of division; they overlap in many places; but this need not disturb the simile. The important distinctions are, that in the toy of our childhood the number of detached pieces was moderate, and each one by itself was not made attractive or interesting, except as it fitted its neighbors and helped make up a whole. In the medical school the divisions have already become a multitude, and each year witnesses further addition and sub-division. And all the while every endeavor is being made, by every instructor, to render each fragment so interesting in itself, that in the plentitude of attractive objects of study, few of the students are likely to keep in mind that the main object of the four years of hard work is not the microscopic study of some or all of the component parts, but that study of the separate departments is preliminary and subsidiary to the study of medicine as a whole, and man as a whole. Medicine, viewed as a science or as an art, is something far higher than a patchwork of its separate departments, precisely as man is far more than the joining together of a certain number of organs. To know the structure and the working of each detached organ and tissue of the body in health and disease is essential, but it is not the sum or the summit of

medical knowledge. Nor is this a sufficient equipment for entering into practice. Nothing less is demanded than the further knowledge of the many obscure, complicated, and most important interrelations between the component parts. With infinite regret we must recognize that to properly impart this sort of knowledge seems to become every year more nearly impossible, mainly, as I think, on account of the increasing numerical preponderance in the teaching staff, of specialists, over the men trained to view things broadly.

How is specialism likely to affect the established lines of medical life in the years which are before us? Many possibilities suggest themselves, of which the most startling is that the family doctor is doomed to practical extinction, soon to be sought for as a rarity in some remote or poverty-stricken region.

In large cities it is very obvious that the children in many wealthy families grow up with almost no experience of medical treatment save at the hands of specialists. It might be amusing to make a list of the various kinds of experts who have in turn looked after the health of a modern heir, combining to do the work which was not so badly done forty years ago by the one general practitioner, who sufficed a family in those days.

This habit, or vice, of employing the services of specialists for even trifling and commonplace ailments, already somewhat established among the rich, is as usual filtering down the scale of income and getting some hold upon that large and fortu-

nate portion of our people who possess neither poverty nor riches. Among them it seems likely to extend, until, as seems inevitable, it is found to be altogether too expensive a luxury. Just what will happen then, is wrapped in impenetrable mystery.

It is needless to discuss the relations of the really poor; this class is now so largely treated at the free clinics and dispensaries, where specialism has its freest scope.

This serious question of the future of the general practitioner has been receiving a good deal of attention during the past year, and has been particularly well discussed in a recent paper by Professor Caillé.* Himself an eminent specialist, though still somewhat engaged in general practice, he expresses without reserve his appreciation of the usefulness of the family doctor, and adds: "I firmly believe that the family practitioner is not doomed to become extinct, and that in due time the people will again elevate him to the position of trusted family counselor." This paper is very interesting, but the remedies which he suggests do not seem to me to reach the root of the disturbance. His article ends with these sentences: "In conclusion, I would venture to express the opinion that all medical men should start as general practitioners. If for any reason whatsoever they find it advisable to practise a specialty, they will be more generously informed and better equipped in every way, by reason of years of

* American Review of Reviews. Apr., 1901.

general practice and experience. I predict that the successful general practitioner of the future will be a *diagnostician*, *sanitarian*, and *minor surgeon*, and will develop into a valuable and conservative general consultant." As an ideal, this is sound and admirable; but I must declare my protest that under the conditions now visible, and those which we foresee to be probable, such a mode of evolution is quite impossible. For, in the first place, it appears to be clear that to the young people now in the medical schools general practice seems to offer no prizes, and that those who are possessed of the capacity, energy, ambition and persistence which will finally make them eminently useful, all these make early choice of a specialty as a career. And, secondly, if it be not true to-day, it will soon become a hard and obvious fact, that it is not possible to gradually shift from general practice into a specialty. A high wall already stands between the two fields, and a man must be on one side of it or the other; he can clear the boundary only by a leap. The family doctors who still survive are sufficiently aware of their own interests not to take the risk of allowing a paying patient to seek special treatment at the hands of anyone who still takes general practice; the temptation to absorb that patient and his family is so strong that to resist it requires a keen sense of honor.

Is it at all remarkable that a specialty should now seem far more attractive to the young graduate than general practice? The inducements seem

to be in every way superior; from the scientific as from the financial side, the prospect is far more alluring; and it is an easy and safe prediction that for many years to come the cream of each graduating class will join the ranks of the specialists.

The general question must be followed a little farther. Assuming that the number of well-qualified general practitioners is to gradually diminish because the natural vacancies are not adequately filled by new recruits, what will follow? Is it likely that the public, or the general medical profession, will be satisfied with the resulting situation? On the contrary, it seems clear to me that a well-defined and imperative demand would soon arise for what is to us almost a new kind of medical man.

According to our present ideas all scientific medical men practically belong to one of the two great groups; they are in general practice, or they are specialists. Assuming that the number of general practitioners is gradually becoming smaller, and of less merit and distinction, and that before many years have passed the specialists will decidedly predominate, then it will become the custom for the public to go directly to some specialist for advice and treatment. This means that the specialist will be selected by the patient or by some non-medical friend. It needs very little experience of disease to appreciate that in multitudes of cases the laity will be unable to rightly decide which particular brand of specialism is required.

Few of us have not felt perplexed on this point, in the presence of an obscure case, for we cannot satisfy ourselves as to the organ which is primarily or most at fault. In such a dilemma specialism does not meet the need, and we have been accustomed to ask counsel of men of the broadest possible knowledge and experience; first, we call upon some one of our neighbors, whose skill we know; in important cases, and as a final resort, we apply to men who have become noted as general consultants, who are often teachers of general or clinical medicine, and visiting physicians in the wards of large hospitals. On reflection it will be found that these invaluable counselors have all had their training in the school of general practice; they have all been family doctors. If this training school falls into desuetude, then we must provide some other source of general consultants and clinicians. Patients must have them, if only to select specialists for them, as did the president of the temple in Egypt, so long ago. Specialists will require them, to guard against the errors inevitable to their own more narrow knowledge. Hospitals cannot do without them, if only to assign patients to the proper wards. Medical schools will need many of such men as professors and instructors, if the purpose holds of turning out really educated graduates. Medical science calls loudly for men of the broadest training, for it is only from such that we may hope to obtain the one most important improvement which our art now requires; namely, a great generalization.

Let some further order be established in the vast mass of recorded facts, and both the study and the practice of our art would become far easier, more reasonable, and more successful.

I hope to live long enough to see the establishment here of a higher medical degree, somewhat corresponding to that of M.D. in England, which shall be a guaranty of very high attainment in a broad range of study, and shall mark the general consultants and clinicians of the future. If it should follow, as in England, that the services of such accomplished men and women shall be sought for ordinary attendance upon the families of those who wish the best attainable skill, and are willing to pay high fees therefor, what an upheaval shall we witness of some notions which prevail to-day! The family doctor will become a luxury which only the wealthy can afford!

The distinction drawn so strongly in England between the functions of a physician and a surgeon, seems to us rather strange and perplexing; but in origin and theory the matter is simple and rather suggestive. The *surgeon* is nothing else than the *chirurgeon;* and he, as the derivation of the word implies, was one whose occupation was a handicraft, or of that nature. Everything involving manual dexterity, from drawing a tooth or opening a vein, to a major amputation, was looked upon as handwork, and therefore on a much lower plane than the purely mental labor performed by a physician. Since most treatment involving manipulation is for lesions which can be

seen or felt, everything on or near the surface of the body or its open cavities came to be considered as in the province of the surgeon. Judged by the ancient standard, it appears that most of the present specialties belong in the lower grade.

Few of us can recall the gradual change in the relation of dentistry to medicine. No doubt some of our oldest members remember when the only dentists were regular physicians; but for at least fifty years there has been so distinct a line of demarcation that we rarely think of dentistry as being included in the domain of medicine.

A very notable tendency of the times is toward increased stress of competition in our ranks as in most other walks of life. No doubt this is going to increase; and indeed there is temptation to predict that it is likely to become a serious calamity before another generation has passed away. All the professions were becoming overcrowded, from causes which are obvious enough, even before the recent great disturbance in social conditions brought about by the growth of trusts and consolidations. The effect of competition in lowering fees and lessening incomes has long been felt and lamented in older communities in Europe, and sooner or later we shall have to face the same unfortunate state of affairs. A little careful study of some of the advertising pages at the back of the *London Lancet* or *British Medical Journal*, will show how pitifully small have become the fees which middle class English people expect to

pay to thoroughly educated medical men. And in almost every number of the same journals may be found letters describing some new club scheme for obtaining skilled advice at even lower rates.

No doubt there will still be room at the top, in the twentieth as in the earlier centuries. The recognized leaders and experts will continue to receive large fees, and many of them; but the rank and file of the profession seem destined to receive on the average a much smaller annual income. I fail to see how any action of ours can prevent or hinder the working of the inexorable laws of nature; demand and supply are sure to settle this question in the end.

One other change of condition must not be overlooked, for it is likely to have very important effects upon the health of coming generations. I refer to the great modifications in the habits, and in the physical and nervous constitutions, of our patients, which the fast increasing predominance of city life is bringing about. There is no time for discussion of this topic, and I can only say that adaptation seems to be a far more leisurely process than the recent and present rapid changes in environment; and that it is reasonable to expect that variation from the types we have considered normal is likely to increase both in frequency and in extent.

In a book once owned by every physician, is to be found the history of a very ancient medical case; older probably than any other to which we

have access. It tells us that a certain Syrian captain, who was a leper, made the journey to Samaria because he had heard that a great healer was to be found there; that he was directed by Elisha to wash seven times in the river Jordan; that with some reluctance he followed the advice, and was healed. After more than twenty-five hundred years this narrative still offers much food for our thought.

It was not a mere coincidence that the healer was also a prophet; priests and prophets were the only healers at that time and place. All disease was then deemed to be owing to the anger of Deity, and to be cured only by supernatural means, through the mediation of the special servants of Deity or of the Temple. We read that "Naaman was wroth and went away and said, Behold, I thought, he will surely come out to me, and stand, and call upon the name of the Lord his God, and strike his hand over the place, and recover the leper." "And his servants came near, and spake unto him, and said, My father, if the prophet had bid thee do some great thing, wouldest thou not have done it? how much rather then, when he saith to thee, Wash and be clean."

The evolution of the race has not yet lifted mankind out of the thought that recovery from disease is brought about by something more mysterious than the forces of nature. We find relics of this old belief everywhere; culture and education seem to affect it little; certainly they never quite wipe it out. The popular acceptance and

approval of every wild new scheme for healing is usually in inverse proportion to the amount of common sense and truth on which the novelty is based.

> These "little systems have their day,
> They have their day, and cease to be,"

but the procession of them never pauses and never ceases. If ever medicine is accepted by the public as an exact science, with methods and results dependent only on natural laws, of course this relic of ancient superstition will cease to exist; but with it will vanish a trait of humanity which has been, and to-day is, far more powerful for good than some of us are accustomed to think. I refer to the patient's personal faith in his doctor.

Of course, when any one of us considers his own relation to his patients, it is obvious enough that their confidence is solidly founded on merit; but if we take a broader view, embracing all the practitioners about us, I think we should agree that, in the case of many of them, there is no such basis of merit. And yet almost every one of these medical men and women, of every school, and of no school;—of every degree of education and experience;—is possessed of clients who firmly believe that he is specially able to cure or relieve their ailments. Viewed in this general way, we must call this faith unreasoning, if not unreasonable; and yet upon it is founded one of our most effectual agents for good.

Every physician, without exception, helps his patients by the expectation of relief which his very

presence brings; and most of us go further, and consciously or unconsciously use the very helpful power of direct suggestion, of a partially hypnotic character. It is curious to note that this faculty and habit of suggestion is the only feature which is common to all who attempt to heal the sick. When the last veil of mystery shall have been torn from our art by the advance of science, I fear that little will be left of our special power of suggestion, and that humanity will be much the poorer thereby.

We observe that the Syrian's leprosy was cured without the use of any drug. We may not all agree as to whether there is any visible tendency toward the disuse of medication; and certainly it would appear that the number of drugs in use, or recommended for use, was never so great as now. On the other hand, we have learned that the virtues of much of the old materia medica were purely imaginary; while as to the value of the new synthetical compounds so pertinaciously thrust upon us, there appears to be wide spread doubt. They are formidable weapons, indeed, but many of them have more than one cutting edge.

The growth of another doubt, of a more radical sort, is also to be noted,—as to whether chronic organic disease is ever cured by drugs, if by cure we mean restoration to the normal state. Some form of compensation seems to be the extent of the benefit which is to be hoped in such conditions. This, at least, can be confidently affirmed, that

in the chronic diseases which figure most largely in the bills of mortality, the use of drugs is decidedly on the wane.

If we are asked to name the specific whose power is most generally granted and least questioned, we should probably at once point to the control which quinine exercises against malaria; yet it seems exceedingly likely that the next century will see less and less use of this great remedy, simply because there will be less need of it;—malaria is going to be prevented. Prevention is a watchword of great promise in the years which are before us; but the promise is to the community in general, and not to those whose incomes depend on the abundance of disease. Our profession prides itself on its constant and eager search for methods of preventing and diminishing disease, and we gain some public credit for our aims and achievements in that direction; but few seem to appreciate the full extent of our unselfishness, and that complete success means professional suicide. This sad result is not yet in sight, even of the prophetic eye we are using to-day.

Returning to the text from which we have wandered so far, we are struck by the statement that it was the little maid waiting on Naaman's wife who called attention to the skill or power of the healer. This little detail has a very modern and familiar sound.

We further read that the grateful Syrian at-

tempted to reward his benefactor with gifts of value, but that the offer was refused. There is nothing modern about this statement. Even that new sect which boasts of its reversion to ancient modes of healing does not carry its reverence for antiquity to such an extreme; it demands the regular fees. Yet to find a modern analogue to the refusal of Elisha, we need only view some of our public and semi-public establishments for medical and surgical treatment. This very day, in almost any city in this state, any person, rich or poor, who is coming down with a fever, or needs a surgical operation, will be received into a city hospital, where he will get first-rate care and treatment. At his departure he will be asked to pay a proper price for his room, board and nursing, but for the professional services not a cent will be charged. What lesson is this likely to teach him and his friends, if not that the municipal supply of medical skill is an excellent and most economical idea, which might be carried further to great advantage.

This practice seems to me altogether wrong, not simply because it diverts much money from the pockets of doctors who deserve and need it, but because it so forcibly teaches a lesson in applied socialism which is very dangerous to the financial future of our guild.

Our text might easily guide us into many other lines of thought, but I mercifully refrain from further turning of the hour-glass, and will simply ask you to observe that this old story of the heal-

ing of the leper has long been known to the whole civilized world. Once, and not so long ago, this was a distinction almost unique; to-day every important medical case or discovery is promptly carried to the remotest regions of our globe. On this opening year of the Twentieth Century, let us not withhold the tribute of gratitude and admiration due to the Medical Press, and particularly to Medical Journalism. Not all of these journals are admirable, and not all are truly scientific; perhaps in some might be suspected a slight bias from commercialism; but as a whole they are doing wonderfully good and useful work, and are to be ranked among the great improvements of modern times. Not all of us are called upon to write papers for them, but every one of us should look upon it as a duty, as well as a privilege, to subscribe to at least one good medical journal.

After all our searching and discussing, the summing up shows but a brief list of plausible predictions. That specialism will increase and competition become more fierce, we may feel as confident as of any future event.

That medical treatment will become more scientific in its basis and methods; that disease will be more effectively prevented; that hygienic agents will largely supplant drugs;—all these seem to be among the strong probabilities.

Very probably the family doctor is about to be eclipsed for a time; perhaps to reappear later in a more glorified aspect.

The darkest cloud which rests upon our future seems to arise from the combined forces of greatly increased competition and the growth of socialistic and coöperative ideas.

We must not forget that the past is the only test by which we judge the future. In his great vision, Dante observed that all the prophets had their heads turned backward. The most accurate inferences we can draw from experience are liable, indeed are almost certain, to be deranged by the entrance of new and unexpected factors. Let us therefore face the future with the hope and confidence which befit men who believe that creation is not without a plan, and that the grand trend of evolution is toward better things.

"New Times demand new measures and new men;
The world advances, and in time outgrows
The laws that in our fathers' days were best."

ARTICLE XXXIII.

## THE SHATTUCK LECTURE.

# THE ALLEGED INCREASE OF CANCER IN MASSACHUSETTS.

By WILLIAM F. WHITNEY, M.D.
OF BOSTON.

DELIVERED JUNE 11, 1901.

# THE ALLEGED INCREASE OF CANCER IN MASSACHUSETTS.

Mr. President and Fellows
of The Massachusetts Medical Society:—

During the past few years attention has been called to the apparent rapid increase of the death rate from cancer, and prophecies of an alarming nature have been made for the future, based on this assumption. With a view to determine what may be the underlying cause of this, the statistics of the disease in this state have been studied, and a comparison made with those of other states and foreign countries; and it is to the results of this that I have the honor to invite your attention this evening.

At the risk of repeating what may perhaps be known to many of you, I will begin by briefly describing the way in which statistics are usually compared and explain a few of the common terms.

The standard by which the mortality of one year or place is compared with another is termed the death rate. By this is meant the number of persons who have died during a year out of a given number who were alive at the beginning. The number usually selected is one thousand, and the rate is easily obtained by dividing the total number of deaths for the whole year by the number of the population alive in the middle of it and pointing off the proper number of decimals. Thus, for example, if it is stated that the death rate of a given year or place is nineteen, it is meant that nineteen out of every thousand persons, without regard to age or sex, alive at the beginning, died during the year.

The rate for any disease may be obtained in the same way by using the number of its deaths instead of the total number from all causes.

Another standard for a separate disease can be obtained by using the proportion of deaths from it during a year to the total number from all causes. This will be called the death ratio, and is best expressed in per cent.

It will be evident at once that the results obtained in these ways are very crude and inaccurate, if the age and sex are not taken into account. For example, the death rate of cholera infantum, a disease restricted to early childhood, would be very misleading if based upon the entire population of all ages.

So for cancer, 99% of the cases of which occur after twenty-five years, it is useless to take into account the population or deaths before that age in estimating the yearly rate and ratio. Then, too, the female sex are generally regarded as more subject to this malady, so that the sexes also should be considered separately.

Unfortunately, in the published statistics of most states and countries these fundamental details are not given, and nothing beyond the crude death rate can be obtained.

In our own state the value of vital statistics was early recognized, and there are quite accurate reports going back to the year 1847 and covering very fully the particulars for the state as a whole. But when it comes to the arbitrary divisions called counties, simply the total number of deaths are recorded, and for towns they are altogether silent in regard to an important disease like cancer, although an entire column of space running through many pages is allotted to the record of those killed in battle or executed, with perhaps the necessity of a single entry for the year.

But unsatisfactory and incomplete as they are in many respects, there is yet much that can be gleaned from them, which it is to be hoped will repay your consideration and criticism.

First, let us look at the death rate for cancer, from 1850 to 1895 by quinquennia calculated from the average deaths of five years, so arranged as to bring the decimal year in the middle, and thus coincide with the year in which the census is actually taken.

In order that the difference may be in whole numbers instead of decimals, the rate is made to apply to a million persons, instead of one thousand. Further, two rates are given, one for persons of all ages, the other for those over thirty years of age, and are as follows:

DEATH RATE FOR CANCER FOR ONE MILLION PERSONS.

| Year. | All Ages. | Over 30 Years. | Year. | All Ages. | Over 30 Years |
|---|---|---|---|---|---|
| 1850 | 153 | 375 | 1875 | 374 | 890 |
| 1855 | 193 | 480 | 1880 | 505 | 1165 |
| 1860 | 257 | 625 | 1885 | 560 | 1295 |
| 1864 | 290 | 650 | 1890 | 605 | 1390 |
| 1870 | 348 | 815 | 1895 | 670 | 1495 |

If a comparison is made between the figures of 1850 and 1895, it is found that the latter are $4\frac{1}{3}$ times greater for the crude and four times greater for the death rate "over thirty years."

Taking the latter figure as the more accurate, it can hastily be said that the death rate has doubled twice in the last fifty years. Assuming the increase to go on at this rate for the next hundred years, doubling every twenty-five, it would give a death rate of 3,000 in 1920; 6,000 in 1945; 12,000 in 1970; and 24,000 in 1995. A death rate more than equal to that of all diseases at the present day. In two centuries the figures would be 590,000 for each million, or, in other words, every second person over thirty would die of cancer during the year; and, in another quarter of a century, the race over thirty years would be practically extinct.

But it will be well to look back once more before we leap so rashly into the future. In the first place, is it cer-

tain that the progression is geometrical—for at its worst it may only be arithmetrical.

The difference between the rate over thirty years for 1850 and 1895 is found to be 1,120, which, if divided equally among the ten quinquennia, would give 112 for each.

If the increase is geometrical, it would be expected that the rate for the first half should be below this average, and for the last above it.

The following are the actual differences for each five year period:

| | | |
|---|---|---|
| 1850 to 1855 | . . . . . | 106 |
| 1855 " 1860 | . . . . . | 145 |
| 1860 " 1865 | . . . . . | 25 |
| 1865 " 1870 | . . . . . | 165 |
| 1870 " 1875 | . . . . . | 75--Average 112 |
| 1875 " 1880 | . . . . . | 275 |
| 1880 " 1885 | . . . . . | 130 |
| 1885 " 1890 | . . . . . | 95 |
| 1890 " 1895 | . . . . . | 105 |

Great irregularity is the marked feature of this table, some of the figures of the first half being higher than those of the second half, and the increase of the last five years of the series corresponds almost exactly with that of the first. From this there is certainly much more proof of arithmetrical than geometrical progression; and if, with this, the horoscope is again cast, it will be found that the rate for 1920 will be 2,060; for 1945, 2,620; for 1970, 3,280; and for 1995, 3,840 instead of 24,000.

Even this number, small by comparison with the other, is high enough, and I must ask you to look still further and see if a redeeming clause can be found for it anywhere.

In estimating the general death rate the failure to properly return or record the deaths is about the only source of error which has to be taken into account. But in the study of single diseases, the errors in diagnosis and the proper

classification of these death certificates also have to be considered. From this the possibility is at once suggested, that the increase, which has been shown above, may be entirely due to more accurate diagnosis and more careful registration.

The question, then, is to find what is the real death rate or ratio for cancer and how closely it corresponds with that obtained from the vital statistics. Where then are there cases which will prove this? And the answer is, in the great hospitals, where the diagnosis has been verified by post mortem examination. Those in our midst are too small and the autopsies too infrequent to furnish very reliable information, so we must turn to foreign countries for it, and in the reports of the k. k. Allgemeiner Krankenhaus, in Vienna, it can be found. There the records of the autopsies have been carefully kept and classified for a series of years.

The cases have been collected by Nothnagel for the years 1870–1881, and 1882–1893, with 1875 and 1885 for the middle point, and for the single year 1865.

The figures and percentages are as follows:—

| | Autopsies. | Cancer Cases. | Death Ratio. |
|---|---|---|---|
| 1865, | 1512 | 122 | 8.02% |
| 1870–81, | 20480 | 1460 | 7.15% |
| 1882–93, | 20358 | 2124 | 10.90% |

They have also been obtained from the reports of the Charite Hospital in Berlin for eleven years.

| | Autopsies. | Cancer Cases. | Death Ratio. |
|---|---|---|---|
| 1875–1885, | 8968 | 569 | 6.35% |

If now these are added to those of Vienna the following is the result:—

BERLIN AND VIENNA.

| | Autopsies. | Cancer Cases. | Death Ratio. |
|---|---|---|---|
| 1870–1892, | 49,806 | 4,153 | 8.3% |

In these large hospitals, although the age is not given, the greater number of patients are adults, and so the ratios are fairly comparable with those for "over thirty years."

From this it is clear that the average death ratio for cancer, based upon carefully performed autopsies, was nearly two per cent. higher thirty years ago than it is to-day in Massachusetts, according to the recorded clinical diagnosis. So that until the death ratio, "over thirty years," reaches more than eight per cent., and then begins to show a decided increase, it is not justifiable to speak of it as due to anything except better diagnosis and more careful registration and classification.

What more careful diagnosis can do is shown from the statistics of the New York Mutual Life Insurance Company, where great care is exercised in verifying the cause of death. The data taken from their published figures is restricted to the ratio for the ages of 50 to 70 years, and is compared with that for Massachusetts in the following table: —

RATIO OF CANCER DEATHS TO TOTAL DEATHS.

| Males (50 to 70 years). | Massachusetts. | 5-year averages. Mutual Life Insurance Co. |
|---|---|---|
| 1880 . . | 5.15 | |
| 1881 . . . . . | | 5.74 |
| 1885 . . | 4.75 | |
| 1886 . . . . . | | 5.65 |
| 1890 . . | 6.10 | |
| 1891 . . . . . | | 5.61 |
| 1895 . . | 6.25 | |
| 1896 . . . . . | | 6.84 |

This shows that the returns are about one-half per cent. higher when made with more care and accuracy. It speaks well, however, for the ability of the average practitioner throughout the State, that the difference is not greater.

To the same conclusion have come Newsholme and King,*

* Proc. Roy. Soc., Vol. LIV.

from their study of the statistics of Great Britain, which show a similar rate of increase to ours, and from a comparison of the figures of an insurance company and of the Frankfort Hospital.

Here this part of the subject may be left, and I will ask you to look back for a moment to see if any of the irregularities of increase, which have doubtless been noticed, are capable of explanation.

The following table gives the death rate of males and females over thirty years for the same period of fifty years, as has been done before for persons.

MASSACHUSETTS. — DEATH RATE FOR ONE MILLION LIVING "OVER THIRTY YEARS." MALES AND FEMALES.

1850—1895.

| Year. | Males. | Females. | Year. | Males. | Females. |
|---|---|---|---|---|---|
| 1850 | 290 | 445 | 1875 | 565 | 1200 |
| 1855 | 342 | 598 | 1880 | 795 | 1500 |
| 1860 | 413 | 822 | 1885 | 835 | 1685 |
| 1865 | 408 | 868 | 1890 | 900 | 1800 |
| 1870 | 532 | 1070 | 1895 | 997 | 1965 |

The first marked irregularity is between 1860 and 1865, where the death rate for males is lower than that for the preceding year. This can well be attributed to the draft the civil war made upon the male population. But it is surprising that the rate for the females should have also been affected by this cause, yet it appears there is nothing like the increase between 1860 and 1865 that there was for the five years preceding or following. It can, however, be explained by the absence of the surgeons in the field, and the faulty registration due to the unsettled state of the community. The effect of the war on the male population is still apparent for the next ten years, since a large proportion of them had perished in the field before arriving at the "cancer age." On the other hand, the females resume their increase with the advent of more skillful attendance and settled conditions.

Between 1875 and 1880 is the largest single gain, and since then it has been gradual, but on a higher level. This era, however, is marked by general introduction of aseptic surgery, and with the hope held out by it cases have been attacked and brought to light which before had found their resting place in vital statistics, under the head of hemorrhage, marasmus, heart failure, old age and the like. And yet for all this improvement, as has been shown above, the limits of accurate diagnosis have not been reached.

So far in considering the rate and ratio all ages have been taken together, but interesting relations are brought out when they are further separated into decades. But as these periods of time pass gradually into each other, the figures can be plotted into a curve to show the gradations between them. If the rates for cancer are expressed on the same scale as is used for the general death rate, viz., one thousand, there will be found such little variation that the line, representing the increase from the earlier to the later years, will be almost a straight one. If, furthermore, it is desired to compare the lines for different years, one with another, the space between them will be so small that they will often apparently coincide. In order to appreciate these differences the lines will have to be magnified, which is easily accomplished by estimating the rate on the basis of one million living at each decade instead of one thousand.

If this is done it will be found that the rate rises slowly from thirty to forty-five or fifty, rapidly to seventy and much less, thence to the end of life. The curves for the males and females have a general similarity. The latter starts higher and increases more rapidly up to seventy, beyond which time it is much less than for the males, so that at the end of life the rates are very close. (Plate 1.)

If now two widely separated years, 1865 and 1895, are compared, the proportion of increase of the two sexes for

each decade can be found. At first glance it seems as if the highest must be in the later years of life for females, as the lines tend to go apart and assume the form of a cornucopia. But if the rate of increase is calculated, assuming the earlier year as one, it will be found that the highest rate is really for the males between 50 and 60 years of age, and the lowest is for them also between 80 and 100.

Another interesting comparison is to find in what decade the female death rate is proportionately the greater when compared to the males. It proves to be between 30 and 40, and then descends quite rapidly to the last decade, where it practically coincides.

In the same way, if it is desired to use the death ratios, their numbers can be plotted in a curve and compared by decades. They emphasize more clearly than the rate curves the high mortality of females during the period from 45 to 55 years of age. The curve of the males follows at a lower level with its highest point at 65 years, while the females' is at a decade earlier. (Plate 2.)

Both the curves of the death rate and ratio can be shown together, and make what may be well termed a "graphic picture." It seems as if each disease has its own characteristic one. This is strikingly shown if those for apoplexy and paralysis, pneumonia and cancer, are placed side by side. In the first, the ratio starts above the rate, ascends in almost a straight line to 80 years, and drops off decidedly in the last decade. The rate swings away from the ratio from 45 years to 65 years, then rapidly rises to cross it and ends very high. (Plate 3.)

The ratio for pneumonia starts much above the rate, reaches its highest point at 45 years and falls somewhat with an undulating curve to the end of life. The rate curve is at first a slowly and finally a rapidly rising one to the end, cutting the ratio at about the same point as the first. (Plate 4.)

The ratio for cancer starts moderately above the rate and rapidly reaches its summit between fifty and sixty years, to fall lower than its starting point at the end. The rate curve much more nearly approximating a straight line, rises gradually, and crossing the ratio, ends at a much lower level than either of the others. (Plate 5.)

Of course the picture will differ a little from those shown according to the relative space allotted to each per cent. of the ratio and five hundred of the rate which in those to be shown after this will be made more equal. It will be seen from them that, go where we will, to the other states of this country or abroad, this graphic picture of cancer will be similar in its general outlines, although some of its details may vary.

I will now pass rapidly in review these graphic pictures of other states and countries, showing wherein they differ from that of our own, first stating that in some only persons are available, while in others both males and females are given.

The comparisons are all made with Massachusetts for the year 1895. The rate and ratio of which are as follows:—

MASSACHUSETTS.—DEATH RATE FOR CANCER FOR ONE MILLION LIVING AT EACH DECADE.

| | MALES. | | FEMALES. | PERSONS. | | |
|---|---|---|---|---|---|---|
| | | | 1895. | | | |
| *Males.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
| Rate, | 153 | 449 | 1302 | 2852 | 3862 | 5025 |
| Ratio, | 1.53 | 3.37 | 5.78 | 6.65 | 4.42 | 2.64 |
| | | | (Plate 6.) | | | |
| *Females.* | | | | | | |
| Rate, | 447 | 1460 | 2897 | 4004 | 5076 | 4952 |
| Ratio, | 4.7 | 11.9 | 15 | 11.1 | 6.87 | 2.85 |
| | | | (Plate 7.) | | | |
| *Persons.* | | | | | | |
| Rate, | 300 | 955 | 2099 | 3428 | 4469 | 3340 |
| Ratio, | 3.1 | 7.55 | 10.37 | 8.87 | 5.65 | 2.77 |
| | | | (Plate 8.) | | | |

MAINE.—PERSONS ONLY AVAILABLE.

| | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 282 | 790 | 1575 | 2770 | 4275 | 4080 |
| Ratio, | 3.6 | 8.44 | 10.07 | 8.95 | 6.37 | 2.8 |

In comparing the ratio, it is found to start a little higher, but its summit is not as high as Massachusetts. The rate curve is also a little lower, and the drop at the end a little more marked.

NEW HAMPSHIRE.—MALES AND FEMALES.

| *Males.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 75 | 298 | 570 | 2041 | 3990 | 4125 |
| Ratio, | 1.1 | 3.18 | 3.60 | 6.12 | 5.26 | 2.7 |
| *Females.* | | | | | | |
| Rate, | 466 | 1280 | 2205 | 2925 | 4160 | 3975 |
| Ratio, | 5.15 | 12.25 | 14.08 | 9.76 | 6.12 | 2.85 |
| *Persons.* | | | | | | |
| Rate, | 265 | 797 | 1435 | 2480 | 4025 | 4075 |
| Ratio, | 3.34 | 8.22 | 9.05 | 7.9 | 5.7 | 2.76 |

Males.—The ratio gives a very irregular curve owing to a singular depression at the decade of fifty to sixty, which is also shown in the rate curve. They are both decidedly below Massachusetts.

Females.—The ratio is very close both in form and height to Massachusetts, and the rate parallels it at a lower level.

VERMONT.—PERSONS ONLY.

| | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 200 | 680 | 1300 | 2190 | 3360 | 3460 |
| Ratio, | 2.75 | 7.64 | 8.88 | 7.66 | 5.50 | 2.61 |

Both curves are somewhat lower, but follow the same general outline as that for Massachusetts.

RHODE ISLAND.—MALES. FEMALES. PERSONS.

| *Males.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 107 | 366 | 1025 | 2240 | 2825 | 4675 |
| Ratio, | 1.06 | 2.70 | 4.3 | 5.17 | 3.92 | 2.70 |
| *Females.* | | | | | | |
| Rate, | 532 | 1453 | 2390 | 3720 | 5160 | 5450 |
| Ratio, | 5.43 | 11.20 | 12.45 | 9.25 | 6.75 | 3.14 |

| *Persons.* | | | | | | |
|---|---|---|---|---|---|---|
| Rate, | 323 | 922 | 1760 | 3020 | 4320 | 2652 |
| Ratio, | 3.27 | 6.92 | 8.20 | 7.30 | 5.37 | 2.96 |

The males are very much lower in both curves until the last two decades. The lines of both are very regular, perhaps fairer than for Massachusetts.

The females are also lower but similar, and the lines are regular.

CONNECTICUT.—PERSONS ONLY.

| *Persons.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 243 | 875 | 1635 | 2618 | 3230 | 3860 |
| Ratio, | 2.70 | 7.45 | 8.30 | 6.20 | 4.35 | 2.51 |

Both curves lower and rather irregular.

NEW ENGLAND STATES.—PERSONS ONLY.

| *Persons.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 282 | 705 | 1835 | 3010 | 4115 | 4360 |
| Ratio, | 3.08 | 6.10 | 9.80 | 8.37 | 5.52 | 2.72 |

(Plate 9.*)

Two quite fair curves, following almost exactly those for Massachusetts, at a little lower level. As in every composite picture the strongest feature predominates, so in this the type is essentially that of Massachusetts, but lowered a little in height from the poorer diagnosis and registration of her sister states.

MICHIGAN.—MALES. FEMALES. PERSONS.

| *Males.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–80 | 80–100 |
|---|---|---|---|---|---|---|
| Rate, | 192 | 410 | 1285 | 2600 | 4325 | 5530 |
| Ratio, | 3.78 | 5.26 | 9.1 | 8.6 | 5.68 | 2.77 |
| *Females.* | | | | | | |
| Rate, | 381 | 1090 | 2340 | 3425 | 5000 | 7560 |
| Ratio, | 5.70 | 13.5 | 17.60 | 12.4 | 6.8 | 3.88 |
| *Persons.* | | | | | | |
| Rate, | 271 | 736 | 1801 | 3050 | 4632 | 6495 |
| Ratio, | 4.81 | 9.06 | 13.05 | 10.55 | 6.17 | 3.30 |

* The x and o in this and the following plates show the ratio and rate for Massachusetts at each decade.

Michigan has but one year of vital statistics which are of value, viz. 1898, and therefore, the curves are based on data from a single year. While the general death rate and ratio is very low, the number of cancer deaths is actually quite high, causing an extremely high ratio.

The males practically coincide with Massachusetts until sixty years when they pass beyond and continue so for the rest of life. The rate curve is lower for females while the ratio is higher except for the last period of eighty to one hundred. A possible explanation of this high rate may be found in a large number of foreigners in the population, if it is true that they are more subject to cancer than native born Americans, as Lyon* thinks has been proved by a study of the statistics of Buffalo. This is borne out by the fact that the proportion of cases of cancer of the stomach and liver is higher in Michigan than Massachusetts (viz. 1.85 to 1.68), from which form of the disease he also claims Germans show a high mortality.

These are the only states which have records in sufficient detail to compare with their own. For New York and New Jersey only the crude death rates can be obtained, which are 6 for New York, 5.12 for New Jersey agains 6.70 for Massachusetts for 10,000 living.

Turning now to foreign countries there are two of which the statistics are complete and accessible, these are England and Austria.

England has been very thoroughly worked up in the Report of the Registrar General and by Newsholme and King, so that I will not trouble you with the details of increase. The latest average date, however, that can be obtained is the year 1885, with which ours of 1895 is compared.

* Lyon, Irving Philip M. D. Cancer Distribution and Statistics in Buffalo. Am. Journ. Med. Science, Vol. cxxi, p. 629.

| ENGLAND AND WALES.—MALES. | | | | FEMALES. | | PERSONS. |
|---|---|---|---|---|---|---|
| *Males.* | 25–35 | 35–45 | 45–55 | 55–65 | 65–75 | 75–100 |
| Rate, | 79 | 297 | 998 | 2299 | 3742 | ·3914 |
| Ratio, | 1.02 | 2.40 | 5.05 | 6.60 | 5.32 | 2.31 |
| | | | (Plate 10.) | | | |
| *Females.* | | | | | | |
| Rate, | 172 | 852 | 2042 | 3368 | 4506 | 4578 |
| Ratio, | 2.32 | 8.07 | 13.55 | 11.85 | 7.53 | 3.07 |
| | | | (Plate 11.) | | | |
| *Persons.* | | | | | | |
| Rate, | 128 | 584 | 1545 | 2868 | 4160 | 4295 |
| Ratio, | 1.70 | 5.12 | 9.05 | 9.13 | 6.42 | 2.38 |

Males. The rate is very close in general, except at the last period when Massachusetts rises considerably higher.

The ratios are also very close, but the highest point in the English curve is sixty years, five years earlier than for Massachusetts.

Females. The rates coincide quite closely and are in general similar although the English is the fairer.

The ratio is lower than Massachusetts but in general on the same lines, but this may be due to the fact that their returns are given for forty-five to fifty instead of fifty to sixty.

This points rather to the fact that the English physicians, as a class, are about ten years ahead of us in diagnosis, and that the registration is also more accurate.

| AUSTRIA.—MALES. | | | FEMALES. | PERSONS. | |
|---|---|---|---|---|---|
| *Males.* | 30–40 | 40–50 | 50–60 | 60–70 | 70–100 |
| Rate, | 171 | 690 | 2065 | 3740 | 3995 |
| Ratio. | 1.94 | 4.90 | 8.20 | 7.42 | 3.33 |
| | | (Plate 12.) | | | |
| *Females.* | | | | | |
| Rate, | 346 | 1075 | 2260 | 3520 | 3980 |
| Ratio, | 3.38 | 8.97 | 10.46 | 7.60 | 3.24 |
| | | (Plate 13.) | | | |
| *Persons.* | | | | | |
| Rate, | 219 | 888 | 2205 | 3620 | 3990 |
| Ratio, | 2.70 | 6.78 | 9.19 | 7.52 | 3.29 |

The rate for males is distinctly higher than Massachusetts, except in the last period which covers from seventy to one hundred instead of from eighty to one hundred years.

The ratio is higher throughout, its summit being a decade earlier, however.

The rate for females is very much lower than Massachusetts and coincides closely with its own male curve.

The ratio is also much lower and appears higher than the rate when compared with the males. This is the only country, so far, where the males are so close to the females both in rate and ratio. The curves are both very easy.

If the death rates of England, Austria and Massachusetts are placed side by side, the fact of the high rate of males compared to females is to be especially noticed, and that Massachusetts has both the highest crude death rate and that over thirty years. In decennial comparisons, it is markedly in excess of Austria, while the intermediate portion of England brings her into the proper place in the curve, as has been seen above.

Finally let us return again to our own country and see what is the difference between the New England states, and if any is shown whether the geographical relationship offers any explanation for it. When this is done a single figure has to be used, and on the whole the most accurate comparison is based on the death rate or ratio over thirty years. It has been found that no essential difference results whichever is chosen, and, therefore, to avoid confusion, only one, the ratio, has been employed. If arranged on this basis the states would appear in 1895 in the following order.

| | States. | Death Ratio over 30 years. |
|---|---|---|
| 1. | Connecticut, | 5.50 |
| 2. | Vermont, | 5.60 |
| 3. | Rhode Island, | 5.88 |
| 4. | New Hampshire, | 5.95 |
| 5. | Massachusetts, | 6.53 |
| 6. | Maine, | 6.75 |

They fall readily into two groups, one between 5.50 and 6, the other between 6.50 and 6.75. Neither group has any geographical relationship between them. When it is further remembered in reality how small the actual difference is, 1.25% between extremes, the assumption is warranted that it can be explained entirely upon statistical grounds without any local cause. Especially as the statistics of the two highest states are regarded as the most accurate of all.

For comparing the different counties of our own state, the total number of deaths over thirty years could be obtained, but the deaths from cancer were given without respect to age or sex. For the state as a whole, however, the number of cancer deaths over thirty had been found to be 96.50 for each hundred cancer deaths of all ages. This figure was used to correct the returns for each county, and thus a close approximation to the true number of cancer deaths over thirty could be calculated. With these two factors then the ratio for each of the countries was worked out for the years 1870 and 1895, as follows:—

COUNTIES ARRANGED ACCORDING TO RATIO OVER THIRTY YEARS.

| | 1895. | 1870. | Rate of Increase. |
|---|---|---|---|
| 1. Plymouth, | 5.12 | 3.41 | 1.47 |
| 2. Hampshire, | 5.83 | 4.12 | 1.45 |
| 3. Barnstable, | 6.08 | 5.26 | 1.14 |
| 4. Worcester, | 6.19 | 4.06 | 1.54 |
| 5. Bristol, | 6.34 | 3.76 | 1.68 |
| 6. Norfolk, | 6.42 | 3.98 | 1.61 |
| 7. Berkshire, | 6.51 | 3.57 | 1.83 |
| 8. Franklin, | 6.67 | 4.44 | 1.50 |
| 9. Middlesex, | 6.72 | 4.31 | 1.56 |
| 10. Suffolk, | 6.79 | 3.98 | 1.70 |
| 11. Hampden, | 6.85 | 3.25 | 2.11 |
| 12. Essex, | 7.28 | 3.83 | 1.90 |
| 13. Dukes and Nantucket, | 8.16 | 4.37 | 1.84 |
| Difference, | 2.16 | 2.01 | |

It also shows the rate of increase from 1870 to 1895,

which in the case of Barnstable County is surprisingly low, and for which there is no especial explanation from its location at any rate.

At first it seems as if there was no geographical relationship between the counties if the numbers are exactly followed. The two lowest on the list are practically widely separated by position and general character of the land. The one bordering on the sea low and sandy, the other inland and high, with ordinary soil.

If, however, the counties are associated by their position on the map, and a new ratio obtained for each group, it is found that the ratio rises progressively from southeast to west for those with about the same population, judging from the number of deaths. The northeastern group, densely populated, and having one and a half more deaths than the rest of the state, shows the highest percentage, but then only one-third of one per cent. more than the western part, with less than a quarter of its population.

COUNTIES BY GEOGRAPHICAL GROUPS.—1895.

RATIO OF CANCER DEATHS. OVER THIRTY YEARS.

| | Total Deaths. | Cancer Deaths. | Ratio. |
|---|---|---|---|
| 1. Plymouth, | 1115.4 | 57.12 | 5.12 |
| 2. Barnstable, | 401.4 | 24.12 | 6.00 |
| 3. Bristol, | 2022.0 | 128.33 | 6.34 |
| | 3538.8 | 209.57 | 6.35 |
| 4. Norfolk, | 1294.6 | 83.17 | 6.42 |
| 5. Suffolk, | 5950.0 | 395.41 | 6.79 |
| 6. Middlesex, | 4638.0 | 312.04 | 6.72 |
| 7. Essex, | 3028.2 | 220.50 | 7.28 |
| | 14910.8 | 1011.12 | 6.95 |
| 8. Worcester, | 2955.4 | 183.13 | 6.19 |
| 9. Hampshire, | 574.4 | 33.51 | 5.83 |
| 10. Franklin, | 413.0 | 27.59 | 6.67 |
| 11. Hampden, | 1305.0 | 89.54 | 6.85 |
| 12. Berkshire, | 784.8 | 51.14 | 6.51 |
| | 3077.2 | 201.78 | 6.65 |

To briefly recapitulate what has been said:—

1. If death from cancer should go on at the apparent geometrical rate of increase of the past fifty years, in two and a quarter centuries every person over thirty years would die from that disease.

2. This rate is probably only arithmetical at its worst.

3. The increase is probably due to better diagnosis and registration, and until the ratio of deaths over thirty years has reached eight to nine per cent., which is shown by autopsies to be the true rate for cancer, it is not justifiable to speak of the increase as inherent in the disease itself.

4. For purposes of comparison with other places or years, a "graphic picture," composed of both the rate and ratio curves, covering the period over thirty years, divided into decades, is the best.

5. Comparison with other states and countries, shows the rate for Massachusetts to be about the same as theirs, with greater variation between the males and females than is the case in Austria, which is remarkable for the correspondence between the two sexes.

6. In the distribution in the New England states, there is no geographical feature that explains the slight variation, which is easily within the limits of better registration.

7. In the state itself there is a slight increase westward for groups of counties of the same density of population. The densest populated part of the state, apart from these, shows a little higher rate.

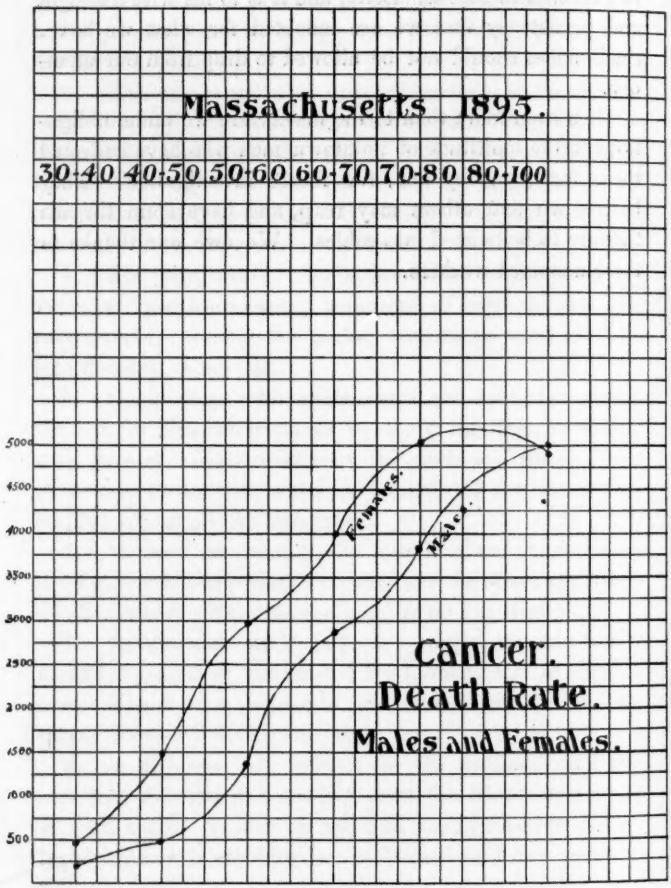
So we will leave the subject for the present, but I feel sure that with this as a starting point the Shattuck lecturer of 1926 should be able to tell you surely whether this alleged increase is anything but statistical as it now seems to be.

In closing, I must ask you to linger a moment that I may recall to your minds Lemuel Shattuck, a distant kinsman of the beneficent founder of these lectures, who must be

classed as one of those who initiated movements which from his time on are perpetual. He may be called the Father of Statistics in Massachusetts, and it is to his wise foresight and persistency that we are indebted for what we have. Such names should not be allowed to drop from our memory.

Then even more than to the master are we under obligations to the multitude of unknown men who have collected these facts, one by one, for future investigation. They have sown that others may reap, and have spun threads that are to be twisted into cables. We owe our thanks to these unnamed workers.

1.


Massachusetts 1895.
30-40 40-50 50-60 60-70 70-80 80-100
5000
4500
4000
3500
3000
2500
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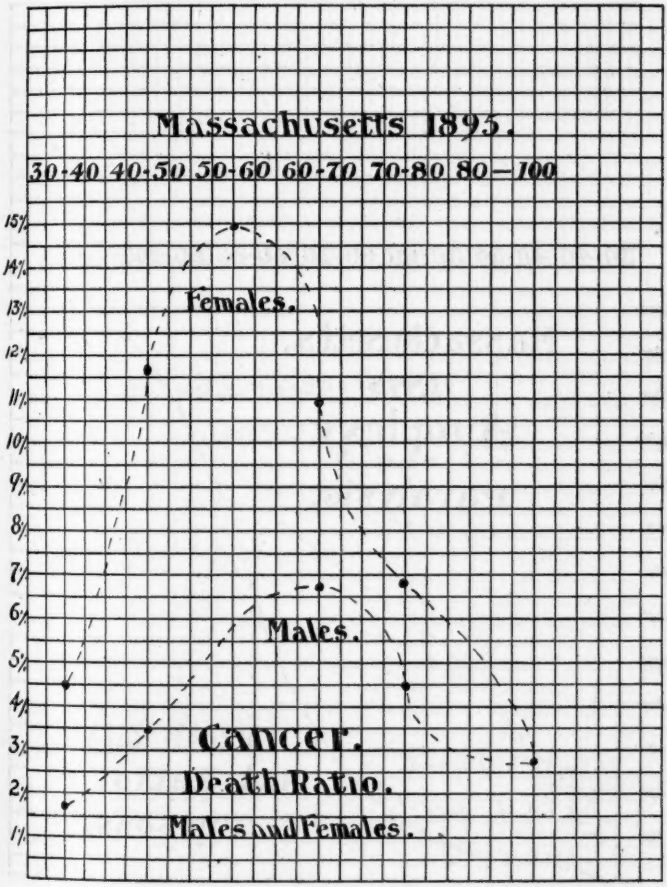
500
Females.
Males.
Cancer.
Death Rate.
Males and Females.

2.


Massachusetts 1895.
30-40 40-50 50-60 60-70 70-80 80—100
15%
14%
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11%
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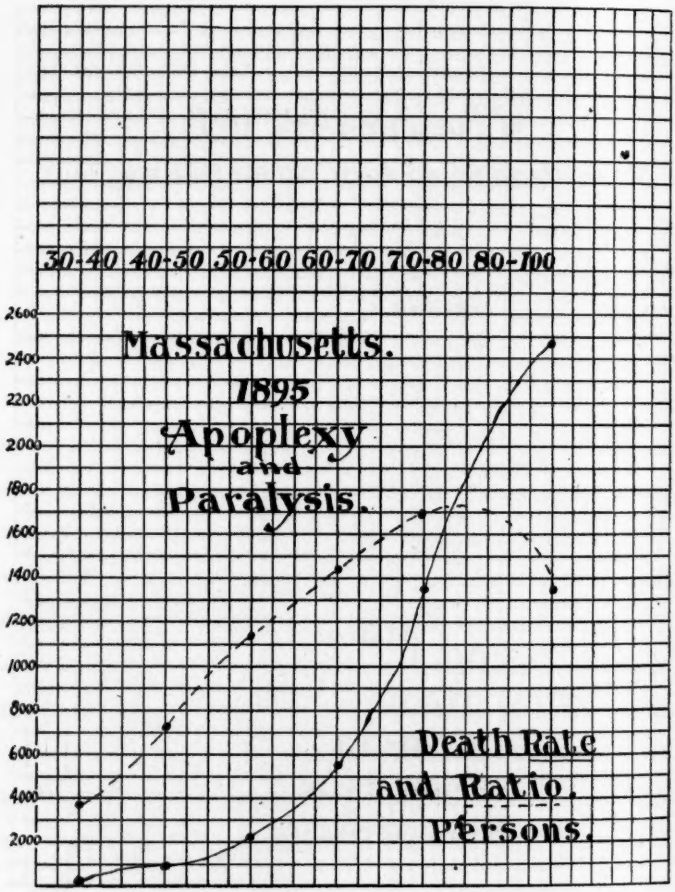
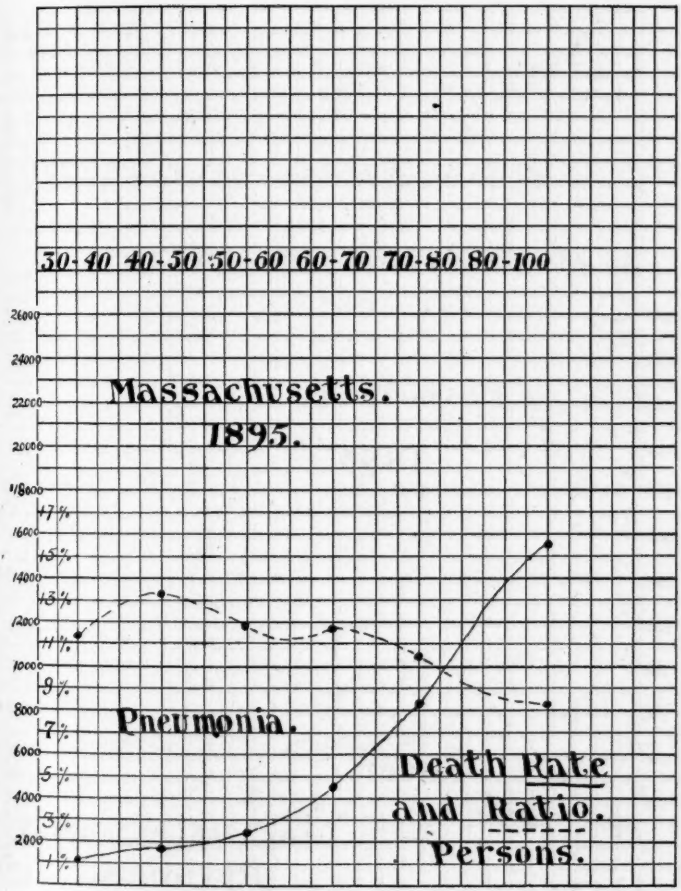
Females.
Males.
Cancer.
Death Ratio.
Males and Females.

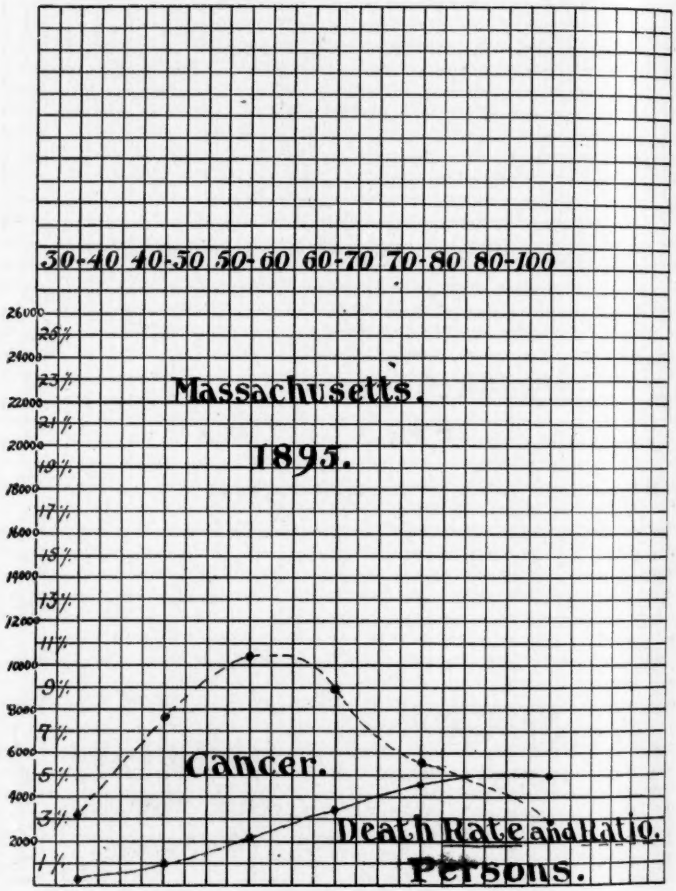
3.


30-40 40-50 50-60 60-70 70-80 80-100
2600
2400
2200
2000
1800
1600
1400
1200
1000
800
600
400
200
Massachusetts.
1895
Apoplexy
and
Paralysis.
Death Rate
and Ratio.
Persons.

4.


30-40 40-50 50-60 60-70 70-80 80-100
26000
24000
22000
20000
18000
16000
14000
12000
10000
8000
6000
4000
2000
47%
45%
43%
41%
9%
7%
5%
3%
1%
Massachusetts.
1895.
Pneumonia.
Death Rate
and Ratio.
Persons.

5.


30-40 40-50 50-60 60-70 70-80 80-100
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5%
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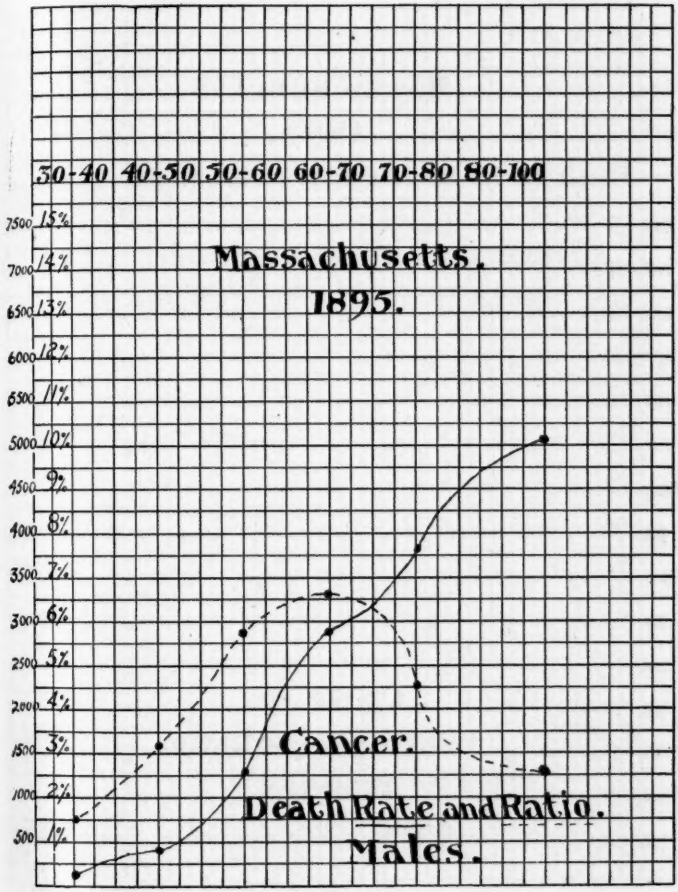
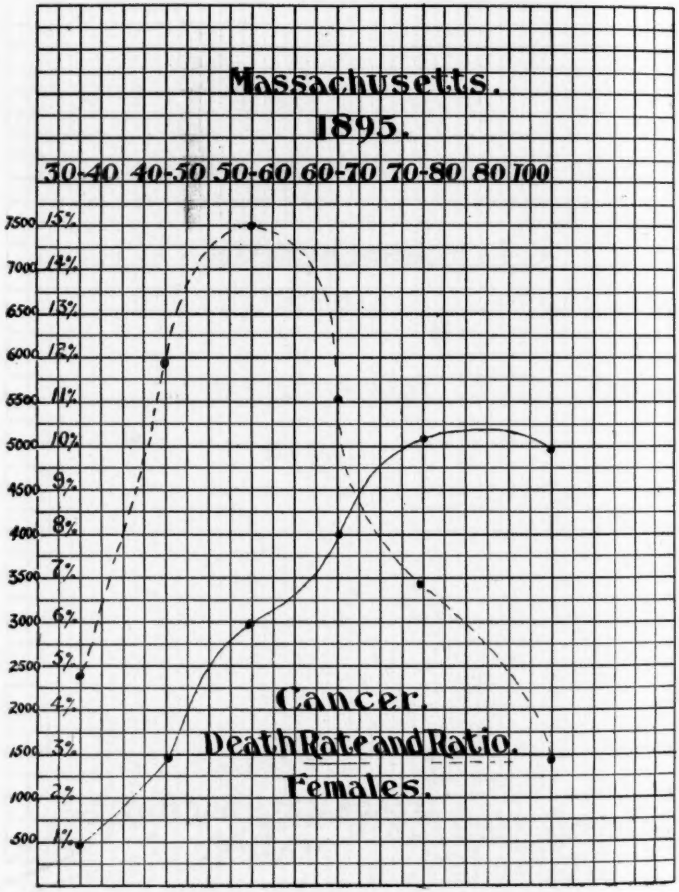
2000
1%
Massachusetts.
1895.
Cancer.
Death Rate and Ratio.
Persons.

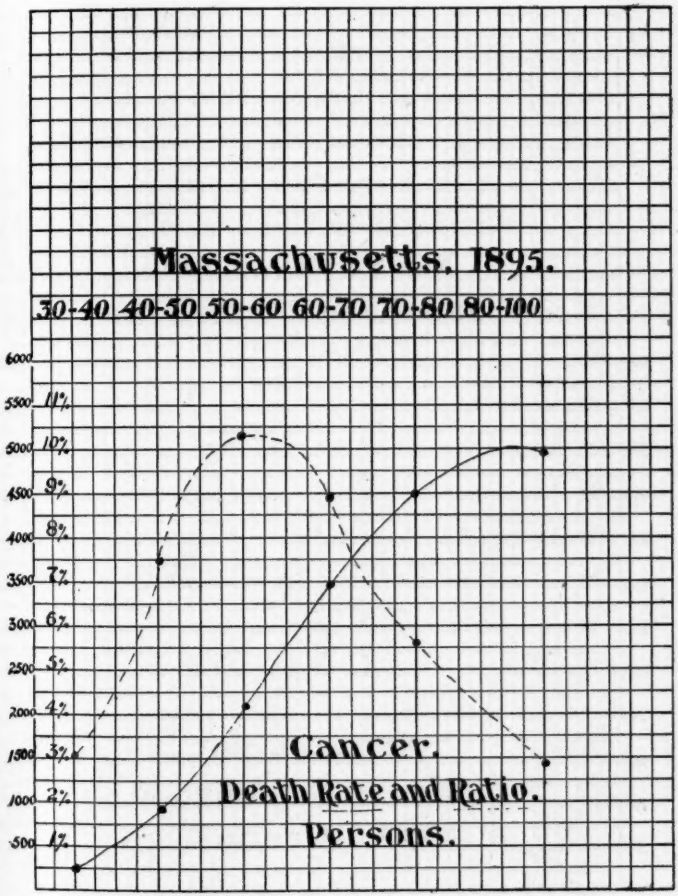
6.


30-40 40-50 50-60 60-70 70-80 80-100
7500 15%
7000 14%
6500 13%
6000 12%
5500 11%
5000 10%
4500 9%
4000 8%
3500 7%
3000 6%
2500 5%
2000 4%
1500 3%
1000 2%
500 1%
Massachusetts.
1895.
Cancer.
Death Rate and Ratio.
Males.

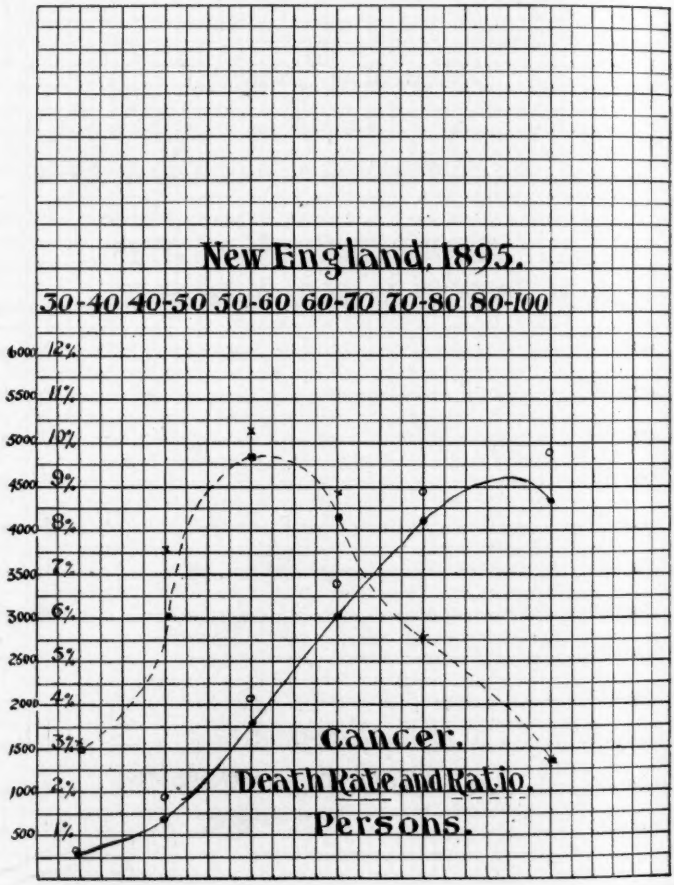
7.


Massachusetts.
1895.
30-40 40-50 50-60 60-70 70-80 80 100
7500 15%
7000 14%
6500 13%
6000 12%
5500 11%
5000 10%
4500 9%
4000 8%
3500 7%
3000 6%
2500 5%
2000 4%
1500 3%
1000 2%
500 1%
Cancer.
Death Rate and Ratio.
Females.

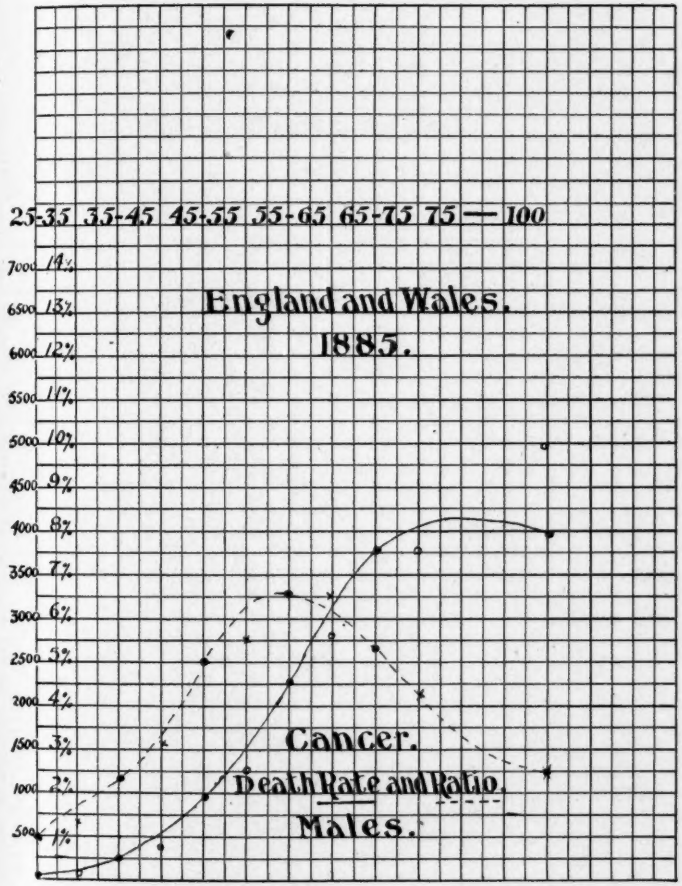
8.


Massachusetts. 1895.
30-40 40-50 50-60 60-70 70-80 80-100
6000
5500 11%
5000 10%
4500 9%
4000 8%
3500 7%
3000 6%
2500 5%
2000 4%
1500 3%
1000 2%
500 1%
Cancer.
Death Rate and Ratio.
Persons.

9.


New England. 1895.
30-40 40-50 50-60 60-70 70-80 80-100
6000 12%
5500 11%
5000 10%
4500 9%
4000 8%
3500 7%
3000 6%
2500 5%
2000 4%
1500 3%
1000 2%
500 1%
Cancer.
Death Rate and Ratio.
Persons.

10.


25-35 35-45 45-55 55-65 65-75 75 — 100
7000 14%
6500 13%
6000 12%
5500 11%
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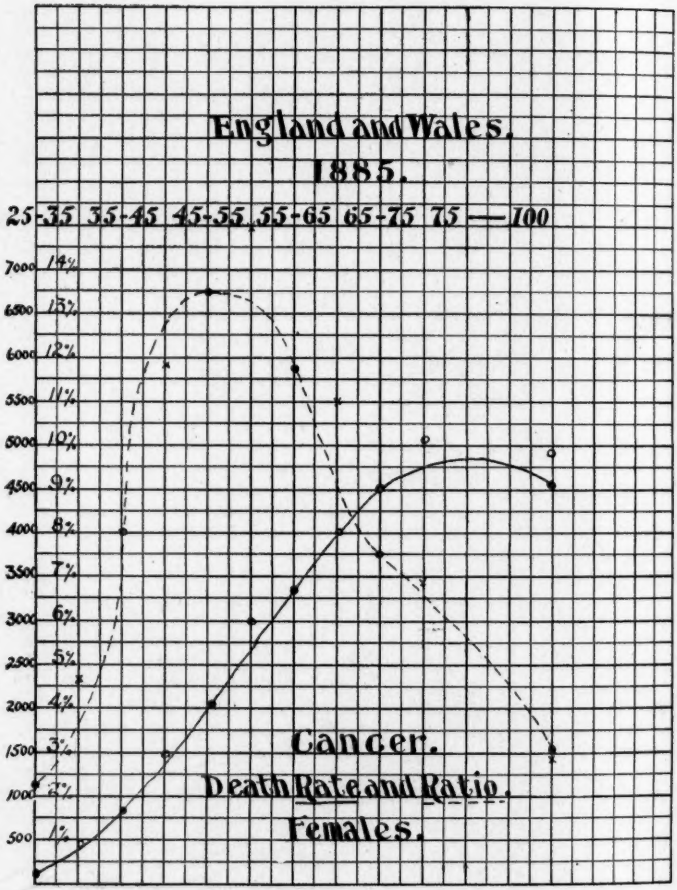
500 1%
England and Wales.
1885.
Cancer.
Death Rate and Ratio.
Males.

11.


England and Wales.
1885.
25-35 35-45 45-55 55-65 65-75 75 — 100
7000 14%
6500 13%
6000 12%
5500 11%
5000 10%
4500 9%
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2500 5%
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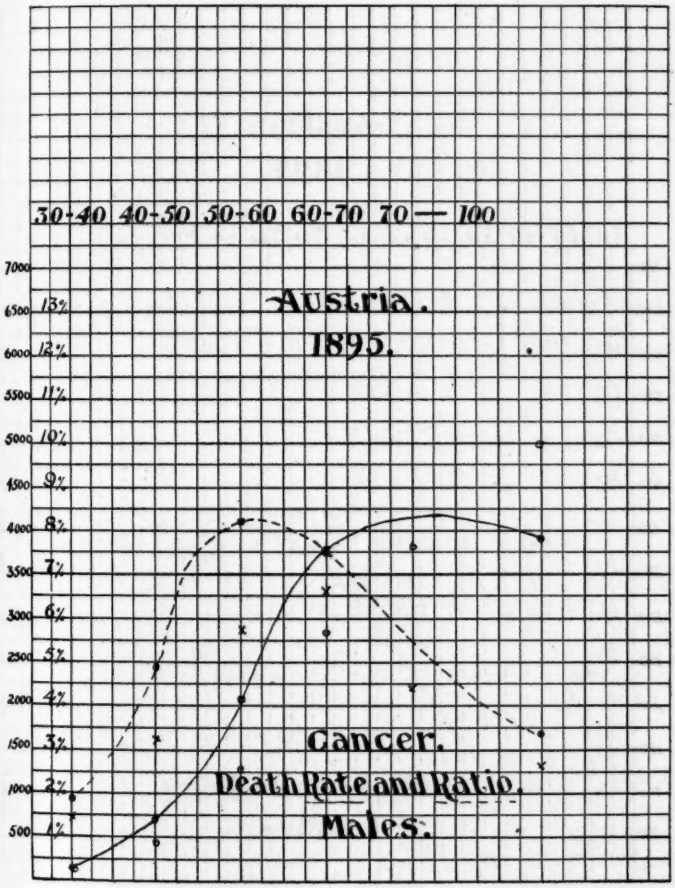
500 1%
Cancer.
Death Rate and Ratio.
Females.

12.


30-40 40-50 50-60 60-70 70 — 100
7000
6500 13%
6000 12%
5500 11%
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4500 9%
4000 8%
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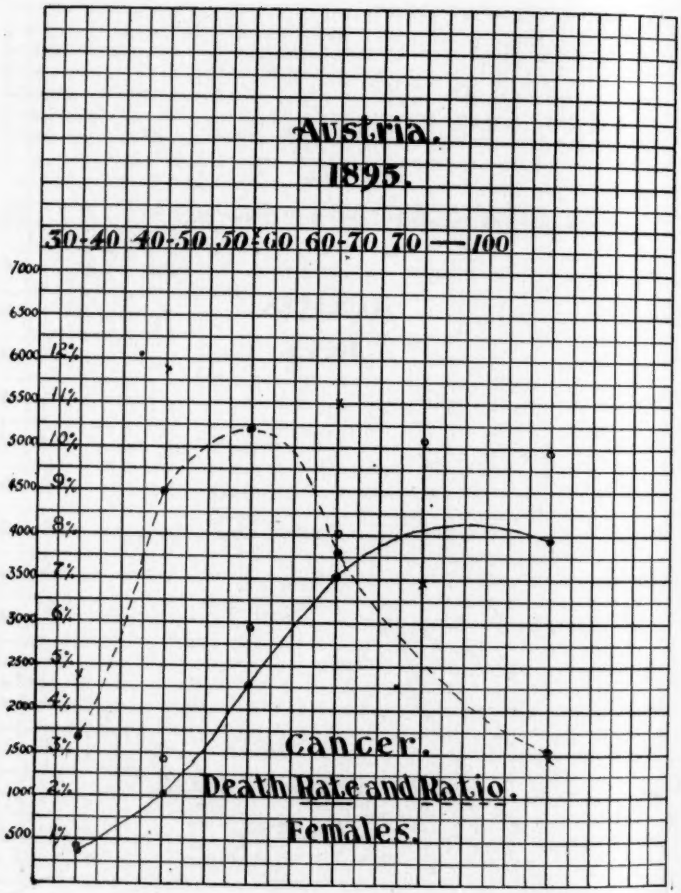
Austria.
1895.
Cancer.
Death Rate and Ratio.
Males.

13.


Austria.
1895.
30-40 40-50 50-60 60-70 70 — 100
7000
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3%
2%
1%
Cancer.
Death Rate and Ratio.
Females.

ARTICLE XXXIV.

# A CRITICAL NOTE UPON CLINICAL METHODS OF MEASURING BLOOD-PRESSURE.

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AND

Mr. C. E. BRUSH, Jr.,

OF BALTIMORE, MD.

From the Physiological Laboratory, Johns Hopkins University.

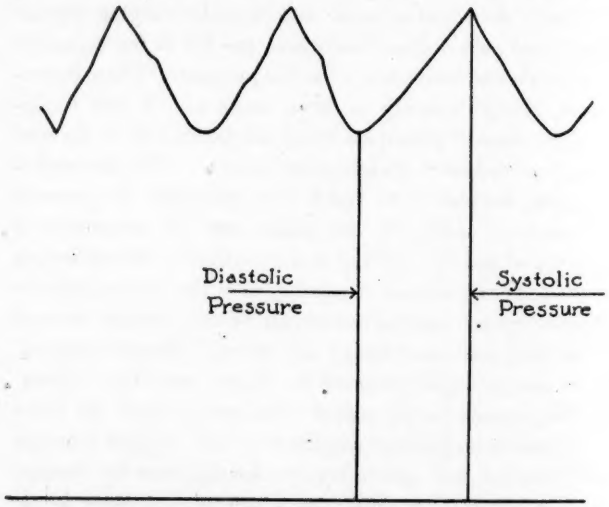
READ, BY INVITATION, JUNE 12, 1901.

# A CRITICAL NOTE UPON CLINICAL METHODS OF MEASURING BLOOD-PRESSURE.

THE recognized importance of a means for determining accurately the blood pressure in man under varying physiological and pathological conditions, has led to the invention of a number of instruments for this purpose. These instruments, though varying in form, make use of two fundamentally distinct principles which are illustrated in the well known methods of v. Basch and of Mosso. The idea underlying the method of v. Basch is to determine the pressure necessary to obliterate the pulse, and his instrument is simply a means for arriving at a quantitative determination of this pressure, instead of relying, as is the case in ordinary practice, upon a subjective estimate of the pressure exerted by the finger in compressing an artery. Mosso's method, based on a principle proposed by Marey, consists in increasing the pressure on the outside of an artery until the pulse wave reaches its greatest amplitude. It is evident that this latter method will give a lower valuation than the former, and it is important to determine which method gives us the more accurate indication of the pressure relations in the interior of the artery.

In considering this problem it is necessasy to bear clearly in mind that the pressure in the arteries varies greatly with each pulse wave. The extent of this variation with the heart beat is greater than is usually supposed. In the carotid of the dog Hürthle found, for instance, that each heart

beat increased the pressure by as much as 100%, from 80 to 160 mm. Hg. In the experiments recorded in this paper, and made by a different method, namely with the use of maximum and minimum manometers, such an extreme variation was found only in the case of abnormally low pressures, produced by cutting both splanchnic nerves. Under what might be called normal or approximately normal conditions, in which the mean blood pressure in the aorta was over 100 mms. of Hg., the increase caused by each heart beat varied between 15 and 58% of the pressure in between the pulse waves. The average of twenty sepa-




rate observations, the details of which are given in the table included in this paper, gave a difference of 33%. That is, the aortic pressure was increased 33% by each pulse wave. It is convenient, following the general usage, to speak of the pressure in the artery at the maximum of the pulse wave as the systolic pressure, and of that at the bottom of the pulse waves, or between the pulse waves, as the diastolic

pressure. According to the average figures given above, the variations in the pressure in the aorta of the dog are represented graphically in the accompanying diagram. In man, with his slower pulse, the variation with each heart beat is probably greater than in the dog. The true mean blood pressure for any artery is evidently the arithmetical mean between the systolic and diastolic pressures in that artery.

It is obvious from these statements that the method of determining the blood pressure by obliteration of the pulse wave, must give, if accurately performed, the systolic pressure in that artery, and this undoubtedly is the result obtained with more or less accuracy by the method of von Basch and the variations of this method used by Potain, Hoorweg, Gärtner, and others. It seems equally obvious, as has been pointed out by Sahli, that the principle used by Marey of ascertaining the pressure under which the artery will give its maximum pulsations, must give the diastolic pressure. A glance at the above diagram shows that when the pressure on the outside of the artery is equal to the constant diastolic pressure on the inside, the arterial walls will undergo the maximum vibration with each pulse.

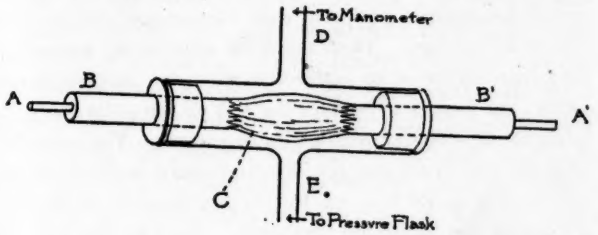
This interpretation of the result obtained by the method of Marey and Mosso was not verified by them upon the living animal, and as the method has been and is being used in this laboratory in various researches, an effort has been made to furnish this experimental proof. Walden published some incomplete results that were not conclusive, and the problem was therefore again tested more carefully. The result, it may be said, is to completely verify the *a priori* probability that the method properly carried out gives the diastolic pressure.

The method used to obtain this result was as follows: The two common carotids were exposed in a dog; one of them, the right carotid, was connected in the usual way

with a mercury manometer writing its record upon a Kymographion. Between the artery and the manometer, however, two Williams valves were so placed as to act, one as a maximum valve, the other as a minimum valve. By means of stopcocks properly arranged, the artery could be thrown directly into connection with the manometer, or through either the maximum or the minimum valve, so that at will either an ordinary record could be obtained or a record of the maximum or minimum pressure. The left carotid was ligated high in the neck, and dissected out for as great a distance as possible. This long stump was then placed in a modified plethysmograph so that its pulsations could be recorded, and the pressure on its outer surface could be raised or lowered at will by means of a pressure flask. In most of the experiments made for the purpose under discussion, the plethysmograph, or sphygmomanometer as it should be called, was made of the same pattern as was used by Walden. It consisted of a conical bag of peritoneal membrane lying in a wide glass tube. The large end of this bag was reflected over one end of the glass tube and held in by a rubber stopper pierced by a hole through which the arterial stump penetrated. The end of the stump of the artery was tied to the small end of the bag, and was anchored by running the cord to the other end of the glass tube, where it was kept in place by the stopper. The pressure flask was connected with the outer glass tubing, as was also a recording manometer. By this means the pulsations of the stump were transmitted to the recording instrument, and as the pressure in the glass tube was raised by elevating the bottle, the loose peritoneal membrane was pressed down upon the artery. As the pressure was raised the pulsations became larger and larger until the maximum was reached, and then smaller as the pressure was still further increased. This instrument and the method of placing the artery in it need not be described in detail, as

it was afterward replaced by a simpler form which had the further advantage that it could be used upon the same artery in which the pressure was being taken directly by the mercury manometer.

The construction of this second form of sphygmomanometer is readily understood from the accompanying diagram:




AA' represents the artery which, after being tied and cut, was drawn through the apparatus as represented, and then, on the peripheral side, either tied down in its normal position and extension, or connected with a manometer to record the internal pressure directly. B and B' represent small glass tubes inserted in each end into rubber stoppers placed in the larger glass tube. The ends of B and B' were connected by a membrane tube C made of peritoneal membrane and with a diameter about three times as great as that of B B'. The large outer glass tube had two openings, D and E; one of these, D, was connected with the recording apparatus to measure the size of the pulse; the other, E, was in connection with a pressure flask. By means of this latter the outer tube and the connections could be filled with water, and then as the pressure was raised the membrane C was forced down on the artery, if need be, until the artery was completely obliterated.

The pulsations of the artery in the sphygmomanometer were at first recorded by a mercury manometer, but it was soon found that with this apparatus there might be much

uncertainty as to the pressure at which the greatest pulse was obtained; the maximum was not clearly marked, especially when the heart was beating rapidly, as after section of both vagi. The difficulty obviously lay in the inertia of the column of mercury which prevented an accurate registration of the quickly recurring pulse waves. The apparatus was therefore changed so as to introduce a Fick's spring manometer, provided with a compound lever to increase its delicacy. Both the Fick manometer and a mercury manometer were connected with the sphygmomanometer, the former to register the size of the pulse wave, the latter to register the pressure on the artery. The procedure adopted was to register the blood-pressure in the right carotid in the usual way on the kymographion, as also the maximum and minimum pressures. Then on the left side the pressure in the sphygmomanometer was raised about 10 mm. at each step, and the corresponding pulse wave was measured with the Fick manometer first, and then the actual pressure by means of the mercury manometer. The pressure was noted at which the maximum amplitude of the pulse wave was obtained, and then again the pressure in the artery on the right side was determined as in the beginning. With the Fick manometer there was never any difficulty in determining the maximum pulse, and the only error of importance in the measurements lay in the fact that the steps by which the pressure was raised in the sphygmomanometer were usually about 10 mms. apart, so that a slight over or under valuation was possible. The results obtained after the method had been perfected were as follows:

| | Observation. | Diastolic pressure in R. Carotid, measured by the minimum manometer. | Pressure at which the maximal pulsations were obtained in the L. Carotid as measured by the sphygmomanometer. |
|---|---|---|---|
| Exper. VII. | A | 156.5 mms. Hg. | 155 mms. Hg. |
| | B | 156 " " | 157 " " |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Exper. VIII. | A | 111 | mms. | Hg. | 112.5 | mms. | Hg. |
| | B | 129 | " | " | 129.5 | " | " |
| Animal bled profusely. | | | | | | | |
| | C | 82 | " | " | 81 | " | " |
| | D | 93.4 | " | " | 85 | " | " |
| Exper. XI. | A | 106 | " | " | 109 | " | " |
| | B | 110.5 | " | " | 110 | " | " |

The measurements made on the two sides were practically simultaneous, and the animal was under such even conditions of anæsthesia that its pressure remained constant through considerable intervals. The agreement in the pressures on the two sides leaves no doubt that Marey's method of measuring the pressure at which the greatest amplitude of pulse is obtained gives very accurately, under the conditions of these experiments, the minimum or diastolic pressure. In the dog the two common carotids spring from the arch of the aorta close to each other, so that on each side what was measured was the aortic pressure at practically the same spot. It is important, however, to emphasize the fact that in estimating the pressure in the sphygmomanometer, measurements were made to the *base* of the pulse wave in the tracing showing the maximum pulse. In the experiments made by Mosso and his pupils with his form of sphygmomanometer it is not expressly stated whether the measurements were made to the bottom of the pulse waves or not, or whether an average was taken between the bottom and top of the pulse wave. The implication, however, is that the measurement was made to the bottom of the pulse wave, as this measurement obviously gives the amount of the external pressure. One might suppose that the maximum pulse would give the full range of pressure from diastolic to systolic, and that therefore measurement to the middle of the pulse wave would give the true mean pressure. Such a supposition, however, is unwarranted, as it is not certain that the mercury column accurately records the full extent of the pressure change,

and moreover the tension of the walls of the arteries and the surrounding tissues will prevent the full extent of the systolic pressure from being transmitted to the water in the sphygmomanometer. The method is adapted to measure only the diastolic pressure.

Mosso's method, as used upon man, consists in putting the ends of four fingers into a plethysmograph or sphygmomanometer of special construction, and determining the pressure, brought to bear on the outside of the fingers, at which the pulsations record their greatest amplitude. While it is quite possible, in this case as in the experiments described above, to use a Fick manometer to record the pulse, the necessary calibration of the instrument introduces both a difficulty and a source of possible error. It is much more convenient to use the mercury manometer to record both pressure and pulsations. In numerous experiments in this laboratory it has been found that if the proper care is taken to have rigid connections throughout, no difficulty is experienced in getting a clear maximum in the recorded pulsations as the pressure is raised. This fortunate fact is due undoubtedly to the relatively slow pulse in the human being. If for any reason the rate of the pulse should be greatly increased, then, as in the dog, there would probably be uncertainty in the determination of the true maximum, and it would become necessary to use some form of recorder more accurate than the mercury manometer.

While the Mosso sphygmomanometer gives an accurate determination of the diastolic pressure upon the naked artery, it remains uncertain whether this same quantitative result is reached when the human finger is used. It would seem that some of the external pressure must be used in overcoming the resistance of the tissues surrounding the arteries, and that therefore the final result reached must be too great by a corresponding amount. What this resist-

ance amounts to cannot be stated, but it is evident that in observations upon the same individual it must constitute a constant factor and therefore disappear as a source of error in comparative measurements. von Basch has estimated the resistance of the tissues at 6 to 8 mms. of mercury and Potain at 10 to 20 mms. of mercury.

Another possible source of error in this instrument has been commented upon by Brush and Fayerweather in a recent paper. It lies in the fact that the arteries in the ends of the fingers are probably small enough to participate directly in vaso-motor changes. If these arteries should be thrown into active vaso-constriction it is evidently possible that the local pressure within them, on the capillary side of the resistance, might be smaller, while general arterial pressure was increased. The authors quoted obtained results in their experiments on the blood-pressure during sleep which they could only interpret in this way, and were therefore obliged to construct a similar instrument to measure the pulsations in the large arteries of the wrist. Hill and Barnard's simple apparatus, which makes use of the same principle as the Mosso instrument, is constructed to take the pressure in the large arteries of the arm just above the elbow, and theoretically should give the diastolic pressure in these arteries. In the experiments made in this laboratory, however, the results obtained with this instrument have been irregular and inconstant, as compared with the results from the Mosso apparatus. This unfavorable result may be due to a want of delicacy in the recording apparatus, arising probably from the fact that much of the pulse is lost by the air-transmission and the elastic walls of the bag and tubing.

The Mosso apparatus as arranged for the fingers is perhaps too complex for ready clinical use, but under the favorable conditions of the laboratory it can be used with facility, and apparently with reliable results as regards the

diastolic pressure. It is generally assumed, however, that it is more desirable to know the mean pressure, and an effort was made to ascertain whether the Mosso instrument can be used for this purpose. The idea was that by raising the pressure in the Mosso sphygmomanometer to the point of complete obliteration of the pulse the systolic pressure would be obtained, and that by taking the arithmetical mean of this and the diastolic pressure, the true mean pressure would be given.

To test this point experiments were made upon the dog's carotid in the following way: One carotid was passed through the sphygmomanometer before described and fastened securely on the other side in its normal extension; the carotid on the other side was connected directly with a mercury manometer provided with maximum and minimum valves. On one side the pressure was raised in the sphygmomanometer until the pulse was just obliterated, while simultaneously the maximum or systolic pressure on the other side was measured directly. It was found that the sphygmomanometer gave a reading of 184.5 mms., while the maximum manometer showed that the greatest pressure at that time in the arteries was only 156 mms., giving a discrepancy, therefore, of 28.5 mms. Part of this may be accounted for by the pressure necessary to overcome the rigidity of the walls of the artery, but according to all measurements this factor could not account for the entire difference. It would seem that even after the artery in the sphygmomanometer is completely obliterated, the pulse waves may still be recorded. We may suppose that each sharp wave of pressure striking against the partially obliterated lumen at the entrance to the sphygmomanometer, or by giving a thrust to the artery, registers some pulsation. In other experiments which were made with the artery lying somewhat loosely in the sphygmomanometer, the pressure necessary to obliterate all pulsation in the instrument was

100 mms. and more above the true systolic or maximum pressure.

This interpretation of the too high result obtained with the sphygmomanometer was strengthened by a series of experiments in which the sphygmomanometer was placed on the left carotid, and the same artery on the peripheral side of the instrument was then connected with a mercury manometer provided with a maximum valve. The pressure in the sphygmomanometer was raised until no pulse got through to be recorded by the mercury manometer beyond. It should be added that to determine the point of disappearance of the pulse the float and pen were removed and the oscillations of the mercury were determined by the eye. As soon as the pulsations disappeared the pressure in the sphygmomanometer was noted and then at once reduced to zero and a reading made of the maximum pressure within the artery by means of the maximum valve.

The following results were recorded:

| | Maximal or systolic pressure. | Pressure in sphygmomanometer at which no pulse could pass through. |
|---|---|---|
| Exper. XI. | 157.5 mms. | 162 mms. |
| Exper. XII. | | |
| Obs. I. | 157 " | 171 " |
| " II. | 160.5 " | 180 " |
| " III. | 158 " | 172 " |
| " 1V. | 158.5 " | 171.5 " |
| " V. | 164 " | 173 " |

In the first experiment the difference between the two readings is 5.5 mms.; in the second experiment, omitting observation II., the difference varies between 9 and 14 mms. This difference is small enough to be accounted for by the extra pressure necessary to overcome the stiffness of the arterial walls. The dog in the second experiment was much larger than in the first, and his carotid was correspondingly larger and thicker-walled. v. Basch has estimated that in the human radial artery a pressure of 1 to 5

mms. Hg. is required to close the empty artery. The results given above indicate a greater rigidity in the case of the dog's carotid. It is quite evident from these experiments that the Mosso apparatus or similar plethysmographic contrivances cannot be used to measure the systolic pressure, a conclusion which has already been announced by Marey and by Colombo. The latter found that in individuals in whom the arteries in the fingers are relaxed and give therefore a large pulse, the outside pressure must be raised to as much as 340–360 mms. to obliterate all pulsations.

The method of determining the arterial pressure in man by obliterating the pulse, according to the procedure employed by v. Basch, Potain and others, should give approximately the systolic or maximum pressure in the artery examined. This *a priori* conclusion has been verified experimentally by Potain with the aid of an artificial schema, and the experiments described above may be taken as a further verification of its substantial accuracy. Under all conditions, however, methods of this kind must give a somewhat supra-maximal result, since some excess above the true maximum is required to overcome the rigidity of the walls of the artery. This factor, according to v. Basch, is small for arteries of the size of the radial or temporal in man, but all criticisms of the method of v. Basch and the various modifications of it proposed, unite in showing that several sources of error combine to give a reading higher than the true maximum, so that Tigerstedt concludes that the error in the readings may vary as much as 32 to 78 mms. Hg., a variation obviously too large for reliable results. These instruments have therefore not come into general use. The recent apparatus devised by Gärtner is, however, so ingenious, and withal so simple and easily applied, that we may assume that it will be used very commonly in clinical work. The principle involved in the instrument is the same as in the method of v. Basch. The end of the finger

is made anæmic by a rubber cap or ring, and the arteries are then blocked completely by a supra-maximal pressure applied to the circumference of the finger through air-pressure. As this pressure is gradually lowered a point is reached at which the blood-stream breaks through and flushes the tip of the finger. Outside the attractive simplicity of the apparatus, the method differs substantially from that of v. Basch in that the obliteration of the blood-current, or rather the moment at which this obliteration is just overcome, is judged by the eye instead of by the sense of touch.

Evidently the theoretical considerations that apply to the v. Basch instrument hold good in the main for the Gärtner. Gärtner himself contends that his instrument gives the mean pressure, but the experimental evidence that he offers is singularly inconclusive. It is quite possible that the blood-pressure taken in the finger by the Gärtner tonometer might approximate the mean pressure, if it should so happen that the blood current did not break through the block promptly for some reason, such as the adhesion of the compressed arterial walls, but such an approximation would be accidental and uncertain. The probabilities on the contrary are that the figures obtained will be if anything higher than the systolic pressure, owing to the action of the same factors that bring about this result in v. Basch's instrument—namely, the elasticity of the arterial walls, the resistance of the soft tissues surrounding the vessels, and the utilization of the velocity increment of the flowing blood. This probability is confirmed by the high figures reported for the arterial pressure in the finger when determined by this method, and also by the comparative experiments of Federn. This latter observer, using both the Gärtner tonometer and the v. Basch sphygmomanometer, reports that the former gave always the higher values. He attributes this result to a vaso-motor effect arising from the process

of making the finger tips anæmic. It seems evident, therefore, that the Gärtner apparatus must be regarded as giving approximately the systolic pressure within limits of error not accurately determined, and that probably the result is always in excess of the actual figure. The experiments recorded above on the naked carotid artery of a dog, in which the outside pressure was determined at the point at which the pulse just ceased to record in the artery beyond the block, corroborates this point of view. It will be remembered that the pressure thus obtained was in excess of the systolic pressure by 5 to 14 mms. according to the size of the artery.

From the facts presented, therefore, we must conclude that of the two principles that have been used, one gives approximately the diastolic pressure, the other the systolic pressure. The arithmetical mean of the results of the two methods should give approximately the mean pressure. If either instrument is used alone it becomes a question as to which result gives the most important indication. To throw some light upon this question, a number of experiments were made upon dogs in which the diastolic and systolic pressures were determined directly, by means of maximum and minimum manometers, under varying conditions that influenced either the heart-beat or vascular tone.

The results are presented in the accompanying table:

| Observation. | Diastolic Pressure. | Systolic Pressure. | Mean Pressure Calculated. | Mean Pressure directly determined by mercury manometer. | Rate per min. | Amplitude. |
|---|---|---|---|---|---|---|
| Exp. X. | *mm.* | *mm.* | *mm.* | *mm.* | | *mm.* |
| I. | 133 | 159 | 146 | 142.75 | 96 | 2.4 |
| II. | 132 | 167 | 149.5 | 144.92 | 96 | 1.75 |
| | | | Severed both Vagi. | | | |
| III. | 174 | 201.5 | 187.75 | 183.87 | 210 | 2 |
| IV. | 172 | 199 | 185.5 | 182 | 210 | 2 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Exp. XI. | | | | | | |
| I. | 100 | 156 | 128 | 127.49 | 114 | 9.83 |
| II. | 108 | 156 | 132 | 129.2 | 120 | 8.4 |
| | | | Severed both Vagi. | | | |
| III. | 161 | 196 | 178.5 | 177.4 | 153 | 5.6 |
| IV. | 165 | 215 | 191.5 | 185.46 | 132 | 6.9 |
| V. | 133 | 160 | 146.5 | 141.69 | 132 | 4.81 |
| VI. | 124 | 158.5 | 141.25 | 145.45 | 126 | 4.85 |
| Exp. XII. | | | | | | |
| I. | 112.5 | 158.5 | 135.5 | 133.35 | 87 | 17.35 |
| II. | 111 | 159 | 135 | 133.9 | 84 | 17.7 |
| III. | 110 | 157 | 133.5 | 133.77 | 84 | 16.87 |
| IV. | 112 | 155 | 135 | 136 | 82 | 13.40 |
| V. | 113 | 155 | 134 | 138.03 | 84 | 17.17 |
| | | | Severed Vagi, and bled profusely. | | | |
| VI. | 129 | 140 | 134.5 | 134.5 | 234 | 1.25 |
| VII. | 128 | 140.5 | 134.25 | 136 | 234 | 1.25 |
| Exp. XIII. | | | | | | |
| I. | 143 | 226 | 184.5 | 182.86 | 153 | 8.5 |
| II. | 144 | 216 | 178 | 169 | 162 | 8.5 |
| | | | Severed both Vagi. | | | |
| III. | 226 | 272 | 249 | 232.5 | 174 | 10 |
| IV. | 188.5 | 258 | 223.25 | 226 | 171 | 10 |
| | | | Severed left splanchnic. | | | |
| V. | 101 | 137 | 119 | 124.75 | 162 | 5.25 |
| | | | Severed right splanchnic. | | | |
| VI. | 37 | 68.25 | 52.62 | 47.33 | 162 | 3.7 |
| VII. | 36 | 70 | 53 | 51.75 | 162 | 3.7 |
| VIII. | 35 | 71 | 53.25 | 53 | 162 | 4 |
| IX. | 38.5 | 74 | 56.25 | 50.82 | 162 | 4 |
| X. | 41 | 79 | 60 | 52.82 | 162 | 3.9 |
| XI. | 45.5 | 78 | 61.75 | 57.24 | 162 | 3 |

An examination of the table seems to justify the following conclusions:

1. The mean blood pressure as usually determined by means of the mercury manometer, corresponds with fair accuracy to the arithmetical mean of the maximum and minimum pressures.

2. A rise of blood-pressure occasioned by an increased heart-beat (section of both vagi) affects the diastolic pressure to a greater extent than the systolic pressure.

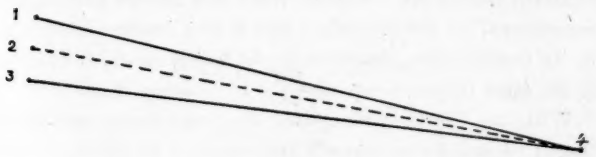
3. A fall of blood pressure occasioned by vascular dilatation (section of both splanchnics) affects systolic and diastolic pressures equally. In the section of one splanchnic, as will be seen from the table, the fall in pressure affected the systolic more than the diastolic pressure, but in this case the heart rate was also affected, whereas section of the second splanchnic did not affect the heart rate at all. The physical effect of a purely vascular change would seem to be shown best under the latter conditions.

4. It is possible for the systolic and diastolic pressures to be affected in opposite directions, as is seen in Exp. XII., in which section of both vagi was intentionally accompanied by a profuse hemorrhage. In this case the mean pressure remained unchanged, the diastolic pressure rising 16 mms. and the systolic pressure falling 15 mms.

It is desirable of course that these experiments should be extended to include a larger number of conditions. As far as they go they indicate that the general trend of arterial pressure, even under extreme conditions of cardiac or vascular changes, so long as these changes are not accompanied by hemorrhage, is given by instruments measuring either the systolic or diastolic pressure. Whether one or another form of instrument is used is a matter of convenience and accuracy. When possible it is most desirable that the results of one method should be checked by the other. As between the Gärtner and the Mosso instruments the former is by far the simpler, but the latter if properly used is probably more sensitive and exact. It is perhaps needless to add that neither instrument can be used successfully for comparative results without careful attention to details.

Many writers have emphasized the desirability of an instrument to measure mean pressures, but to the present

authors the special importance of such an instrument seems doubtful. In the circulation the pulse wave disappears in the capillaries. At this point in the circulation, therefore, the systolic pressure, diastolic pressure and mean pressure all coincide. The relations of the three factors may be represented in principle by such a schema as the following:




[1] Systolic pressure.
[2] Mean pressure.
[3] Diastolic pressure.
[4] Pressure in the capillaries.

For comparative purposes it would seem to be a matter of indifference which of the three factors is known at any given point in the arterial system, provided the method used can be shown to give reliable results. For purposes of analysis and accuracy it would be much more desirable to have parallel records of the variations in systolic and diastolic pressure than a single record showing simply the mean, even if the latter measurement was possible. In our ordinary laboratory experiments we follow the latter plan, to be sure, but we would gain a much clearer insight into the details of vascular changes under varying conditions if the orninary manometer record was supplemented by maximum and minimum tracings.

In the use of the Mosso apparatus on man the experience acquired in this laboratory shows that the following precautions are perhaps the most important for good results: 1. All connections between the recording manometer and the sphygmomanometer should be rigid and inextensible to prevent loss of pulsations. 2. The fingers should be warmed before placing them in the apparatus, or, as is our

usual custom, the first water driven through the apparatus to remove the air should be very warm. 3. If the heart beats are so rapid as to give an uncertain maximum, it is necessary to employ a spring recorder of some kind. 4. The first results upon any individual are usually too high, owing doubtless to the psychical factor. Lower and more constant results are obtained when the subject has become accustomed to the procedure and is in a relaxed condition. 5. In comparative observations the hands should always be at the same height to equalize the hydrostatic factor.

With the Gärtner tonometer the precautions just mentioned (4 and 5) are equally important. In addition, care must be taken in comparative observations to place the pressure ring on the same part of the finger; when over the joints of the phalanges, lower pressures may be obtained than when between the joints. Some attention must be paid also to the initial pressure in this pressure ring,—it is well in successive observations to start with initial pressures that are approximately equal. Finally, those who make serial observations upon human beings with these instruments, under normal or pathological conditions, must bear in mind the important fact, noted in a preliminary communication by Dr. Clelia D. Mosher, that in both men and women there are periodic oscillations of arterial pressure.

ARTICLE XXXV.

# PRACTICAL BLOOD EXAMINATION.

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READ JUNE 12, 1901.

# PRACTICAL BLOOD EXAMINATION.

Among the special methods of the study of disease which have been developed in this recent era of laboratory methods, one of the most important and practical in the aid which it lends us in the understanding and diagnosis of our cases is that of the examination of the blood.

The usefulness of this method as a clinical procedure rests first upon the value of the knowledge obtained by its application in connection with the diagnosis of many of the diseased conditions commonly encountered in general practice; and second upon the simplicity as regards technique of the methods involved in this application, a simplicity which fits the examination perfectly for routine use in clinical work. Experience of ten to twenty years in blood examination has established for us very definitely the scope of this method in the diagnosis of disease, and has given us a set of very practical methods for its accomplishment.

The scope of practical blood examination established by use at the present time is, stated briefly, as follows:

I. The determination of the existence or non-existence of the condition of anæmia, and if such a condition is present, of its severity and type, including in the latter determination the diagnosis of pernicious anæmia.

II. The determination of the existence or non-existence of the condition of leucocytosis, and, if such a condition exists, of its extent and type, this including the diagnosis of the conditions of myelogenous and lymphatic leukæmia.

III. The determination of the presence or absence of blood parasites, this including the diagnosis of the condition of malaria, that of filaria sanguinis hominis and that of relapsing fever.

IV. The determination of the existence of definite serum reactions in the blood, as, for example, the Widal test for typhoid.

V. The determination of presence in the blood of bacterial organisms, *i. e.* of septicæmia.

The accomplishment of these determinations is the scheme of practical blood examination. Barring the tests for bacterial infection, the determinations of septicæmia and the serum reactions, which involving as they do some practical knowledge of bacteriology and of culture methods form a special branch of blood examination, this whole scheme of practical blood examination giving the diagnosis of anæmia, of leucocytosis, and of the presence of blood parasites, and comprising to-day nine-tenths of all clinical blood work, may be accomplished by the employment of two very simple methods of examination. These methods are (1) the estimation of the hæmoglobin, and (2) the examination of a stained specimen of blood. Other methods, as the examination of fresh specimens or the enumeration of the corpuscles by special counting methods, may confirm and add to our knowledge as to the condition of the blood, but the simple procedure mentioned, consisting of the performance of these two methods, will give us our aid to diagnosis, where such aid is obtainable from blood examination, without the use of further methods.

The procedure of these two methods is as follows:

For the estimation of the hæmoglobin we have a very simple method known as the Tallqvist method. For its employment we need as apparatus simply a needle to draw the blood and one of these specially prepared books known as the Tallqvist hæmoglobin books.

This book contains specially prepared porous paper, which will soak up the blood, and a color table. This color table contains ten color plates, the top one representing the color obtained by soaking the special porous paper of the book in a blood containing the normal amount or 100% of hæmoglobin. The next plate is a facsimile of the color obtained by the use of a blood containing 90% of the normal hæmoglobin. Each succeeding plate represents the color effect of a blood of 10% less color richness than the one above it, until the last plate represents a blood of 10% hæmoglobin.

To perform the test it is necessary to prick the ear or finger so that the blood flows without pressure, and draw off a drop or two in the porous paper, allow the blood to dry, and then compare the specimen at the moment of dryness (not earlier or later) with the color table. If the color corresponds to that of the 100% plate, then the hæmoglobin content of the blood is normal; if to the fifth plate, it is 60%, and so on.

This test tells us at once and without further testing whether or not a condition of anæmia is present.

For the preparation of a stained specimen there are necessary a half dozen thin glass cover slips ($\frac{7}{8}$ square slips are the best), a needle, an alcohol lamp or gas burner, or some chemical fixing material, as absolute alcohol, for fixing the blood, a staining mixture for the staining, and a microscope with an immersion lens for the examination.

Thin spreads are made upon the cover slips by dropping one containing a drop of blood upon a clean slip and sliding them apart after the blood has spread. These spreads are dried in the air and then fixed. A simple method of fixing is to hold the cover slip in nippers for two minutes above a flame, at a point just too hot to hold the hand for any length of time. By practice the correct point with an alcohol lamp or gas burner can be determined. For heating a number

of specimens a copper plate kept at a constant temperature over a flame, or a constant temperature oven may be used. The heat required in the oven or on the plate is 110 to 120°C. for five minutes' time. The fixed blood is then stained by an appropriate stain.

The object of staining blood in clinical work is to enable us first to differentiate the varieties of leucocyte present, second to discover the malarial parasite if present, and third to distinguish any nucleated red corpuscles present. The leucocytes of blood have been found to be of three varieties according to their staining reactions or affinities. One form takes by preference an acid stain in its protoplasm, another a neutral stain, and the third form a basic stain. So to differentiate our leucocytes we must subject blood to all three kinds of stain, or to a staining mixture containing all three stains. This is what is meant by an appropriate stain. Such a staining mixture we possess in the commercial stain known as the Ehrlich triple stain or three color mixture. This mixture, composed of two acid stains, acid fuchsine and orange G, and one basic stain, methyl green, contains at the same time an active acid, neutral, and basic element, staining the protoplasm of the acid leucocytes, the acidopiles or oxyphiles (eosinophiles) red or golden red, that of the neutral leucocytes or neutrophiles, lilac, and that of the basic leucocytes or basophiles, blue. The elements of the protoplasm taking these special stains are fine granules scattered through the basal substance which remains unstained. The nuclei of all the leucocytes stain blue with the basic elements of this stain. The red corpuscles take a golden color with this stain, the nuclei of any nucleated red corpuscles, if such are present, take the blue basic stain. In order to get constant and definite results with this Ehrlich method of staining it is found necessary frequently to modify it somewhat by the application of a second purely basic stain to the blood subsequent to the regular Ehrlich

mixture. This modification brings out much more definitely than the simple Ehrlich mixture, the basophilic granules in the protoplasm of the basophiles and makes the differentiation of the three types of leucocyte a much simpler process than in the original method. It also makes the differentiation between nucleated red corpuscles and the round nuclear leucocytes—the lymphocytes and other basophiles much more definite, and finally stains the malarial parasite, a result which the simple Ehrlich method often fails to accomplish.

This modified Ehrlich method of staining therefore attains all the objects of blood staining as above enumerated.

The process of staining is as follows:

The blood properly fixed is stained for two minutes in an Ehrlich three-color mixture. A useful formula for making up this mixture is the following:

| | |
|---|---|
| Ehrlich-Biondi-Heidenhain; three color mixture | 1.5g. |
| Acid fuchsine . . . . . | 0.4g. |
| Absolute alcohol . . . . . | 2cc. |
| Water (distilled). . . . . | 15cc. |

The specimen is then washed and stained for one-half second to two seconds in Loeffler's alkaline solution of methyline blue.

The specimen is then washed, dried and mounted in balsam. By the use of this method of blood examination, a process necessitating, save for the microscope, the simplest forms of apparatus, and taking for its application in simple cases fifteen minutes, in complex ones perhaps an hour, we can determine in a given case the existence or non-existence of a leucocytosis and its type, the presence or absence of blood parasites, and if anæmia be present, as proven by the hæmoglobin test, its type.

The diagnosis of the type of anæmia by this examination of a stained specimen is a simple matter. We have in the various diseases which have anæmia as an associate, from

the point of view of the blood finding, a considerable variety of anæmias. Among these, however, no variety is characteristic of any one special clinical condition as distinguished from other conditions having anæmia associated, save one, namely, that variety associated with the symptom complex or diseased condition known as pernicious anæmia. So for practical work it is essential to determine but two types of anæmia in the blood characteristics, the pernicious type and the non-pernicious. In the condition known as pernicious or idiopathic anæmia we have a type of blood finding peculiar to this condition, not found, with certain definite exceptions to be mentioned, in the anæmias of any other known condition.

The recognition of this type is therefore of much importance, first for diagnosis of the condition, and second because, since prognosis is always, with a single exception to be mentioned, bad where such a finding is present, it gives us a definite line for prognosis in our case. This diagnosis is made as stated by the examination of a stained specimen of blood.

In a specimen of normal blood stained by the modified Ehrlich method, the red corpuscles appear as circular biconcave discs of a diameter averaging 7.5$\mu$ and a yellow color marked in the perimeter and faint or absent in the central area.

Their characteristic in normal blood is that of uniformity. The shape is, save where an artefact of spreading, round or slightly oval; the size practically uniform to the eye though actual variations in diameter from 6 to 9$\mu$ are present, the color yellow, and the whitish central areas of the same regular size in all, save where the corpuscles are crushed in spreading when no white centre appears.

In the blood of a case where the hæmoglobin test shows anæmia we may find in the stained specimen great variation from this uniform normal picture. We may find that the

corpuscles are much paler than in the normal, the white centres of the corpuscles occupying a much greater proportion of the corpuscle. This condition is known as achromia. In some anæmias, as that associated with the condition known as chlorosis, this is often the only abnormal characteristic present in the stained specimen. We may find variation in the shape or size of the corpuscles from the normal character. Thus we may see balloon or triangular forms, forms much larger or smaller than normal. The picture becomes one of diversity rather than uniformity. This condition of distortion in size and shape is spoken of as poikilocytosis.

Also in severe anæmia we may find present red corpuscles containing nuclei—forms known as blasts. These blasts may be of the size of normal red corpuscles, when they are called normoblasts, or they may be larger than normal when they are called megaloblasts. They stain like the red corpuscles in their protoplasm. The nuclei are as a rule single, very compact in structure, and stain blue or bluish black by the Ehrlich method. The presence of these blasts means a severe anæmia. The megaloblasts are of more serious significance than the normoblasts.

All these changes and abnormal forms mentioned may occur in both the pernicious and the non-pernicious anæmias. Each and every abnormal form, poikilocytes, macrocytes (large corpuscles), blasts both normoblasts and megaloblasts, which is found in pernicious anæmias, may be present in the very severe anæmias following cancer or hemorrhage. The more severe these conditions are the more the blood approaches the pernicious type of anæmia. The combination of the abnormal characteristics is however always different in the two types, giving a special picture to pernicious blood not found elsewhere.

This special feature of the blood of pernicious anæmia is the presence of an excess of large nucleated red corpuscles

or megaloblasts. To diagnose the condition, therefore, we search our stained specimen for blasts, counting separately all normoblasts and megaloblasts. If more megaloblasts than normoblasts are present the condition is pernicious anæmia. If the normoblasts are in excess, or if no blasts whatsoever are found in a sufficient search, we diagnose the anæmia as of the non-pernicious type.

In addition to this essential characteristic, the blood of a typical case of pernicious anæmia has other features which are more or less peculiar to this condition and hence of some importance in the study of the case.

The first is the existence of a general tendency to increase in the size of the red corpuscles the so-called high-volume index. The existence of this condition is observed in the stained specimen. Large red corpuscles do occur in severe anæmias of all types. A marked average increase, however, such a condition that the space necessary to hold a given number of corpuscles is distinctly larger than it would be with normal blood, is rare outside of pernicious anæmia. The condition is not, however, present in all cases of pernicious anæmia. The existence of this high-volume index may be demonstrated also by the centrifugal method of blood examination.

The second peculiar feature of pernicious blood is the high-color index. This characteristic is determined by the comparison of the estimation of hæmoglobin and that of the number of red corpuscles.

It is a common but not constant feature of pernicious anæmia.

A diminution in the number of red corpuscles, usually a reduction to less than two million per cubic millimeter, is also a common characteristic of this condition. It is not constant, however, and may occur in other forms of anæmia.

The establishment of these additional characteristics is of use in aiding in the complete understanding of our case.

The diagnosis is made, however, simply by the examination of the stained specimen, where we can determine the excess of megaloblasts and also where present the existence of a high-volume index.

I have said that this peculiar blood finding of pernicious anæmia was limited to this disease with certain definite exceptions.

A similar blood picture is presented by cases of infection by intestinal parasites as anchylostoma duodenale and bothriocephalus latus. In these conditions the anæmia disappears and the case recovers upon removal of the cause. This fact must be borne in mind in deciding upon diagnosis and prognosis in a case with such a blood finding.

In some cases pernicious anæmia bloods have been reported as associated with atrophy of the stomach; also a few cases have occurred in association with nephritis and other chronic conditions. These facts simply tend to enforce the opinion which is that generally accepted in regard to pernicious anæmia, that it is but a particular form of secondary anæmia due to special causes and conditions for the most part unknown, and for the most part in the light of our present knowledge unremovable.

Anchylostoma is one cause of this severe type of anæmia which is known and is curable.

This opinion is further encouraged by the fact that severe anæmias, with cancer or hemorrhage or other causes, as they become more advanced, tend to approach more and more nearly to the pernicious type. The volume index becomes greater, though not as a rule over the normal, the color index nearer 1, and megaloblasts appear, though not in excess of the other blasts.

The diagnosis of a leucocytosis is made by a study of the stained specimen of blood, prepared as above described.

The study of blood has shown that there are present in normal blood from 4000 to 10,000 leucocytes per cubic

millimeter of blood; that is, a proportion of 1–500 to 1–1000 red corpuscles. The leucocytes making up this total are of three kinds, which are differentiated on the basis of their staining reactions with a triple stain. These three forms are basophiles, cells taking a blue stain in their protoplasm; neutrophiles, cells taking a lilac stain in their protoplasm; and oxyphiles, cells taking a golden red stain in the protoplasm.

These three forms of leucocyte are present, each in definite proportions in normal blood. Thus in any given blood, of one hundred leucocytes 20 to 35 are basophiles, 60 to 75 neutrophiles, and ½ to 5 oxyphiles. So that in a cubic millimeter of blood we may have, according as our number of leucocytes is high or low (4000—10,000), from 800 to 3500 basophiles, 2400—7500 neutrophiles and 40 to 500 oxyphiles.

A leucocytosis is an actual increase in the number of one or more forms of leucocyte, basophile, neutrophile, oxyphile, over the maximum normal limits. That is, if we have more than 3500 basophiles or 7500 neutrophiles in a cubic millimeter of blood, we have a leucocytosis. This leucocytosis is named after the kind of cell increased. Thus, if it is the basophiles which are increased actually, we call it a basophile leucocytosis; if neutrophiles, a neutrophile leucocytosis; if oxyphiles, an oxyphile leucocytosis. If more than one form is increased over normal for convenience we name the leucocytosis after the form which is most increased proportionally to its normal standard, though such a condition is in reality mixed leucocytosis.

To make a diagnosis of a leucocytosis, then, we must determine whether or not the actual number of any form of leucocyte is increased over the normal maximum for this cell. The simplest method of the determination of such an increase, if it exists, is the determination of the presence of an increase in the total number of leucocytes present. If

such an increase exists we know that there must be actual increase in the number of at least one special form of leucocyte—that is, that we have a leucocytosis.

For most cases in clinical work this determination may be made by the examination of a stained specimen. Some practice in the study of stained specimens of normal blood and of the blood with various degrees of leucocytosis will enable one to determine with fair accuracy in a given case whether the number of white corpuscles present is within the normal limits or distinctly increased. Where the number is high-normal or slightly increased, this method may leave one in doubt. In such cases, if necessary, the point can be determined by a count of white corpuscles by the Thoma-Zeiss method.

As a rule, where a leucocytosis of sufficient importance to have a distinct bearing on the diagnosis of the cases is present, it is of sufficient degree to be determined by this quick method of estimation of a stained specimen. The determination may frequently be made at a glance. In other cases it is necessary to count the contents of a given number of miscroscopic fields and compare the results with normal results. It is perfectly true that a leucocytosis may exist without an actual increase in the total number of leucocytes. One form of leucocyte may be actually increased and the other forms decreased. Such a condition can be diagnosed only by a white count, plus a differential count and actual computation of the number of each kind of leucocyte per cubic millimeter. The necessity for such a procedure is suggested by the character of the case and the appearance of the blood in a stained specimen. In a great majority of our cases, we can and do estimate definitely, for purposes of clinical work, whether or not we have a leucocytosis by the examination of the stained specimens alone. This point determined, we determine its type by a differential count of the leucocytes, naming the leucocytosis for that form of leucocyte most increased proportionally.

The existence of a leucocytosis may also be determined by examining a fresh specimen of blood. This method, however, like the white count, tells us nothing of the type. The importance of the determination of the type of leucocytosis must not be underestimated. The common form of leucocytosis is the neutrophite leucocytosis. It is this form of leucocytosis which accompanies suppuration; many of the infectious diseases, as pneumonia, scarlatina and diphtheria, and many inflammatory conditions. The oxyphite leucocytosis is characteristic of trichinosis, of some skin affections. Its presence often suggests the diagnosis of one of these conditions. The basophile leucocytosis is characteristic of lymphatic leupæmia. It is found in children in pertussis and rachitis. The myelocyte leucocytosis is, as stated, characteristic of myelogenous leupæmia.

And for this determination, both of the existence of a leucocytosis and of its type, we use the method of the stained specimen.

In addition to the forms of leucocytosis mentioned, we have in certain diseased conditions another form known as a myelocyte leucocytosis. This is a condition in which we have an afflux of large numbers of an abnormal form of leucocyte known as myelocytes into the blood. These myelocytes are neutrophilic or oxyphilic cells, resembling in morphological character normal leucocytes, but differing from the normal neutrophiles and oxyphiles in the fact that they are non-amæboid and have as a consequence round instead of the many-shaped nuclei of the normal cells.

They are differentiated from the normal cells in a stained specimen by their staining characteristics and this morphology of their nuclei.

These cells are never present in normal blood. They may occur in a variety of diseased conditions. When few in number and unassociated with increase in the number of

leucocytes their appearance has no special known significance. When they appear in connection with a great increase in the number of leucocytes, in such numbers that it is evident that the leucocytosis is, in considerable part, due to their presence, we speak of the condition as that of a myelocyte leucocytosis. It is this condition which characterises the blood of myelogenous leupæmia. A recognition of this leucocytosis is therefore important in connection with the diagnosis of this disease.

The parasites which we may find in the blood in disease are the malarial parasite, the filaria sanguinis hominis and the spirillum of relapsing fever. The appearance of these parasites in the stained specimen of blood is as follows:

In a specimen stained by the modified Ehrlich method the malarial parasite appears as a blue body lying within a red corpuscle. The appearance of the organism is dependent upon its age and type. If fully formed, dark pigment will appear scattered through its substance. If in an early stage of development it may appear in the form of a blue signet ring or as a simple sphere without pigment.

Where we suspect the presence of the malarial plasmodium we ordinarily look at a fresh specimen of blood, since the organism is more satisfactorily determined while alive and moving in the blood. Where it is impossible to get to the bedside of the patient at the proper time, the examination of stained specimens will, however, suffice for diagnosis. As stated, we may use the modified Ehrlich staining method for staining malarial organisms. A more satisfactory method for this special purpose is, however, the Jena method.

The Jena staining mixture consists of a mixture of eosin and methylene blue put up in the following manner:

| | | |
|---|---|---|
| 1% | Aqueous Solution of Eosin, | 100cc. |
| 1% | " " " Methylene Blue, | 100cc. |

Allow to stand 24 hours. Filter. Dissolve the precipitate in methyl alcohol to saturation.

This method gives very excellent stains of the malarial parasite. The method of fixing by alcohol preserves the histological structure of the various bodies to be stained more perfectly than the fixing by heat.

This staining method may also be used in place of the Ehrlich method for regular blood staining after one has studied the characteristics of blood by the Ehrlich method. It has the advantage over the Ehrlich method of fixing the blood at the same time that it stains it, giving a constant method of fixing in many ways superior to the fixing by heat. The objection to the method for general work lies in the fact that it contains no neutral staining agent and thus differentiates but two forms of leucocyte—the oxyphile and basophile. It does, however, stain the cell, which would be a neutrophile by the other method, a much fainter red than the oxyphile. So that, possessed of a knowledge of the distinction between these cells, gained from the use of the Ehrlich method, the student has no difficulty in working with this method.

In addition to these methods of practical blood examination, established by experience as useful, there are some recently-developed methods of equal practicability for application but lacking the testimony of extended experience in regard to their usefulness.

Among these, the most important and promising is the test for iodophilia. Recent investigations have shown that if pus, due to the action of the common pyogenic bacteria, be subjected to a special iodine solution, consisting of iodine, 1; iodide of potassium, 3; water, 100; gum arabic q. s. to make syrupy in consistency, certain of the pus corpuscles will show granules in their protoplasm, taking a brownish red stain with the iodine. If blood from a patient suffering from such suppuration be stained with this same mixture, it is found that certain of the neutrophilic leucocytes take this same iodine stain—perhaps 10 in 100 of all leucocytes.

This phenomenon is called iodophilia. It is said to occur in conditions of suppuration, except those due to tuberculosis, and in croupous pneumonia. It is also present in severe anæmias and in the leukæmias.

As stated, the usefulness of this method is still sub judice. If future experience shall show that it does occur in the stated condition exclusively and constantly, it will offer us an unequalled means of determining the existence of suppuration in a given case, or of distinguishing pneumonia from other infectious conditions.

Other blood phenomena, of recent discovery, which can be determined by simple methods of observation, are the granular degeneration of the erethrocytes in lead poisoning and the peculiar staining characteristics of the blood of diabetes mellitus. As the diagnosis of these conditions can, however, be made more definitely by simpler methods of observation, these tests are not to be considered as necessary parts of practical blood examination.

This, then, with the addition of the serum reaction tests, is the present scope of the blood examination, practical for the regular practitioner in his routine work.

The special application of the knowledge gained by the use of these methods to diagnosis is part of the study of clinical medicine and must be taken up in connection with the study of particular cases. Its general application can readily be understood from the above review.

Exact knowledge in regard to the presence or absence of anæmia in a given case is always to be desired, and where it can be obtained by so simple a procedure as that described in our method should always be secured.

The presence of a leucocytosis is one more fact to help us in deciding what a febrile attack may have as a cause, in diagnosticating pneumonia or suppuration or in ruling out typhoid or malaria.

The determination of its type may make our diagnosis of leukæmia or lead us to the diagnosis of trichinosis.

The finding of parasites is of course of absolute diagnostic value.

My object in this paper has been simply to demonstrate to you the method of procedure necessary for the accomplishment of these diagnoses and to impress upon you the extreme simplicity of the work, in the hope that this examination may be more commonly used in general practice.

ARTICLE XXXVI.

# THE GENERAL CHARACTER OF THE PROBLEMS OF PUBLIC HEALTH BACTERIOLOGY.

By HIBBERT W. HILL, M.D.,
OF BOSTON.

READ JUNE 12, 1901.

# THE GENERAL CHARACTER OF THE PROBLEMS OF PUBLIC HEALTH BACTERIOLOGY.

THE problems confronting public health bacteriological laboratories are so numerous, diversified and intricate that even a brief enumeration of them would involve reference to almost every department of bacteriology.* I will try,

* As examples: —

*Diphtheria.*—Is there a relation between types of K. L. and of clinical diphtheria? Is there a relation between morphology and virulence? Do virulent diphtheria bacilli lose virulence in the throat or nose in time, and does such loss correspond with morphological changes? Does the diphtheria bacillus (as is widely taught) degenerate or otherwise change in morphology during the convalescence of the patient? Are certain forms of bacilli (Hoffman's, etc.), known to be widely distributed in healthy throats, true diphtheria bacilli or are they closely allied to them, perhaps as a saprophytic form of the true pathogenic bacillus? Are they capable of gradual transition under favorable conditions to the pathogenic form? Can diphtheria be contracted otherwise than from previous cases?

*Tuberculosis.*—To what extent do true tubercle bacilli occur in healthy throats or noses exposed to the disease and not exposed? Do forms resembling tubercle bacilli occur which are liable to be mistaken for them on examination? What is the relation of the smegma bacillus to the tubercle bacillus? What is the distribution of the smegma bacillus? What is the relation of both of these to the "timothy hay bacillus?" Can tuberculosis be contracted from other sources than previous cases (directly or indirectly)? What are the relations of bovine and human tubercle bacilli?

*Typhoid Fever.*—What is the relation of this bacillus to the colon and paracolon groups? What are the relations of the Widal reaction to typhoid, paratyphoid, paracolon and colon infections?

These are a few of the questions coming under but three of the pathogenic bacteria dealt with in public health work. While some of the questions are qualitative in the given form, calling merely for yes or no as an answer, it is probable that many of the answers cannot properly take this form but must be quantitative in character; thus diphtheria bacilli may perhaps lose virulence *sometimes* during convalescence, *sometimes* not; and it may some day be possible to tell in what percentage this loss occurs if at all, and what are the contributory circumstances. These questions re-

therefore, rather to point out their general characteristics, confining myself to one or two illustrations only. To set them forth clearly, the relation of public health bacteriology to other fields of bacteriology must first be outlined.

### THE RELATION OF PUBLIC HEALTH BACTERIOLOGY TO OTHER FORMS OF BACTERIOLOGY.

To begin with, bacteriology as a science deals always with the potentialities of certain minute particles of living protoplasm. These potentialities make themselves evident by the reactions, physical, chemical, biological, which they produce upon their surroundings and therefore bacteriology is less a direct study of the bacteria than it is a study of their environments and the relations of the bacteria to them. The difficulty of bacteriological investigation is due largely to this fact, while the importance of a full knowledge of these potentialities is so great in so many directions that notwithstanding the difficulties involved, it is essential that the knowledge be achieved.

In chemistry and physics, the fields of the academic research worker along purely theoretical lines are sharply distinct from those of the worker in applied chemistry and physics, but in bacteriology, there exists as yet in this country no definite class of men who deal solely with theoretical bacteriology. Almost all the bacteriologists of this country are engaged in applications of the science to some practical end.

Applied bacteriology finds expression at the present time in four more or less distinct fields.

Industrial bacteriology is concerned particularly with the

quire wide investigation and the obtaining of so many individual results as to permit of *quantitative* deductions therefrom. They are not new problems, qualitatively considered, but much remains to be done on the qualitative side alone, while the quantitative side has been as yet scarcely attempted in many cases. The quantitative side is of the greatest possible hygenic importance, as a moment's consideration will show.

action of bacteria as they affect certain industries of economic importance. While indirectly of hygienic value, the immediate object of industrial bacteriology is the placing on the market for consumption of articles (chiefly foods) which are better or more acceptable for the attention paid to their bacterial relationships.

Pathological bacteriology (using the term pathology in its ordinary, rather restricted, sense) is occupied chiefly with the relations of certain bacteria to the animal body. While essentially analytic, it has ultimately a hygienic bearing, pointing out not only the processes of bacterial diseases but also the best methods of interfering with and so overthrowing them.

The two remaining fields of bacteriology are directly hygienic. These are water and sewage bacteriology on the one hand and board of health bacteriology on the other. The water and sewage bacteriologists are concerned principally with those very important life processes of bacteria which come into play in the pollution of water and the purification of sewage. Their work brings them in contact with sanitary engineering particularly and, in this country at least, the typhoid bacillus is practically the only distinctively pathogenic form with which they deal.

The board of health bacteriologist is concerned more intimately and directly with hygienic work in its general sense. Although dealing much with bacterial diseases, his field lies less in the study of existing disease processes than in the study of questions connected with the origin, transmission and suppression of diseases. The pathological bacteriologist considers the relations of pathogenic bacteria to the sick; the public health bacteriologist considers the relations of pathogenic bacteria to the well. The public health man deals with the crude materials; the pathological bacteriologist deals with the finished product.

These general fields of bacteriology are of course more

or less interrelated, and much light, direct and indirect, may be obtained by each of them from the others. But each necessarily has problems peculiar to itself, which bear particularly on it and for the solution of which it cannot look to the others. Bacteriology as a whole owes much to pathology. Without the attention drawn and the impetus given by the study of the relations of bacteria and disease, bacteriology would not yet have reached its present position. But the bacteria of disease are a small and specialized group, the proper elucidation and appreciation of the relations of which depend ultimately on work in lines more general and more connected with nature at first hand than pathological bacteriology in the ordinary sense can supply. It seems probable, therefore, that the bacteriology of the future will advance in correlation with the advances of preventive medicine, and will reach its highest development in these and in industrial lines.

### THE GENERAL FORM OF THE PROBLEMS.

Ten years or so ago, bacteriological laboratories as adjuncts to Boards of Health were almost unknown in this country; to-day every large city and many considerable towns possess them. They vary in size and equipment from one small room, where a local practitioner spends an hour or two each day examining diphtheria cultures and sputum, up to the large buildings of the New York Board of Health, one of which, at least, is designated as wholly for research. Since much of the work of these laboratories is diagnostic, one problem constantly pressing upon all of them is to send out early and accurate reports. This is a very important matter if the laboratory is to meet the wants of the Board of Health and of the physician. But to attain this ideal of "early and accurate reports" involves many contributory problems in itself. The promptness of

reports depends largely upon executive arrangements but their accuracy depends upon complete knowledge and perfect methods. Unfortunately our knowledge is incomplete and our methods imperfect. We are therefore constrained to acquire more information, and to improve methods, or, failing this latter, to determine the imperfections of the present methods with a view to their indirect elimination.

The examination of diphtheria cases by culture for diagnosis and for release furnishes such excellent examples of both of these problems, that I shall describe some of its features as illustrations of the general character of certain of the questions now pending—questions which apply to every form of diagnostic work and indeed to all bacteriology.

In diphtheria diagnosis, in the majority of cases, the bacteriologist can say that the culture submitted to him shows or does not show the presence of diphtheria bacilli. But not infrequently, certain forms of bacilli are found which, while morphologically perfectly familiar, must still be considered as belonging to those forms, the true relations of which to the diphtheria bacillus are not yet determined. To this day, there is evidence and authority of the highest on both sides of the question, one affirming and the other denying that these particular forms of bacillus are forms of the diphtheria bacillus. Bacteriologists have struggled with this difficulty for years, but it still remains a difficulty. Renewed attention has been directed to it of late by Wesbrook of Minneapolis. To indicate the importance of its practical bearing, it is only necessary to point out the principal results to be expected from its solution.

If these bacilli are not forms of the diphtheria bacillus, many communities in which they are now regarded as such will be justified in releasing convalescent diphtheria patients perhaps a week on the average earlier than at present. This means one hundred years in time more liberty for five thousand cases of diphtheria. Much trouble in dealing

with institutional epidemics will be eliminated and a large number of the persons now quarantined as infective under such circumstances may go free. The diagnosis of diphtheria also will be made somewhat more accurate, although fortunately the error from these forms is small in this particular.

If however these forms be diphtheria bacilli, if they be true infective agents, present ideas concerning the epidemiology of diphtheria will need very radical revision, since probably one-third of the members of most civilized communities have these forms of bacilli in their noses or throats, or both. Present methods of diagnosis and present procedures regarding isolation and release will require very radical remodelling.

One may foresee, however, other possible solutions. It is conceivable that these organisms are diphtheria bacilli in the classificatory sense, but are nevertheless non-virulent so that they need no consideration from the diagnostic or hygienic standpoint. Perhaps some of them, indistinguishable morphologically from certain forms of true diptheria bacilli, may not be diphtheria bacilli in any sense. It is conceivable that only a few of them are infective, most of them not. But without speculating any further, it is evident that the solution of this problem, legitimate, necessary, pressing as it is, is not a matter to be arrived at off hand. It brings up questions of morphology, of polymorphism, of evolution, of degeneration, of classification, of nomenclature, and many others which can only be solved with much time and patience. It involves a reconsideration of the basic conceptions of bacteriology, perhaps their reconstruction in some directions.

It may seem at first glance strange that a problem so simple as this one apparently is should be so difficult of solution. In the older and more developed sciences, similar problems could very probably be readily settled by the application to them of previously established factors.

But in bacteriology, the factors are not yet fully established —indeed they are less firmly established now than heretofore. In the older days, a bacterial species was a definite entity; to-day we shrink from the use of the word species at all because we do not know what it means,— or that it means anything. For example, the typical colon bacillus and the typical typhoid bacillus present so many distinctive features that one is amazed to think that their differentiation should ever have given rise to any difficulty. But a complete chain can be constructed in such a manner that, with the typical typhoid bacillus and the typical colon bacillus at opposite ends, the intermediate links composed of atypical typhoid bacilli and atypical colon bacilli taken consecutively show very little difference from one to another. A similar state of affairs is probably true of other species and almost surely of the diphtheria bacillus. Indeed, we are by no means sure that the diphtheria bacillus is a bacillus in the old sense, nor exactly what we mean by the word bacillus itself. These are not merely interesting academic questions; they are essential matters relating to every-day life and practice.

Such investigations necessarily carry the investigator deep into the foundations of bacteriology. It is at this point that all bacteriologists meet, however diverse their special fields. Upon each worker, industrial, pathological, hygienic, it presses, for new problems cannot be readily solved with the foundations shaking under our feet, although that shaking is not from decrepitude, but from the movements of reconstruction. Such problems press peculiarly on the hygienic bacteriologist because his are problems peculiarly connected with the ultimate factors of health and disease.

If, however, our knowledge were perfect, our methods would still be imperfect. The methods of chemistry and physics are imperfect, unavoidably so. The absolute can-

not be reached. Chemists and physicists having realized this long ago, eliminate the difficulty, so far as they may, by determining just how accurate their methods are, and then allowing for the slip—the working error. Yet for some reason, one is constantly called upon to combat a widespread impression that bacteriological methods differ from those of every other science and art in being themselves infallible. In diphtheria diagnosis, for instance, the accuracy of a report depends first on obtaining a representative culture from the patient, then in having it properly inoculated, properly grown and properly examined. Slips at some points in this chain occur to an extent which makes an error, if only one culture be taken, and consisting in failing to find the bacilli in the culture when they are really present in the throat, of from five to ten per cent. of the cases which finally prove positive. The working error in release examinations is much larger, reaching an average of thirty per cent. and constituting the sole reason for the requirement of two consecutive negative cultures for release. Even the presence of the diphtheria bacillus does not always mean that the patient is suffering from the disease, since a certain number of healthy unexposed persons and a larger number of healthy exposed persons present them.

Another problem, neither executive nor technical, evolves itself from these considerations; the impressing of the fact that working errors exist, and their natures, on those who use these methods, so that they may allow for them and more intelligently employ the reports they receive. In the older days, a bacteriologist's report required only direct and simple translation into terms of a corresponding clinical diagnosis. This form of empiricism gave, it is true, a high percentage of successes; but like other empirical procedures, it was apt to leave the empiricist high, dry and puzzled at unexpected and usually most inopportune times. Since both our knowledge and our methods are imperfect, it is the part of wis-

dom to take these imperfections into account. No one can make a proper and intelligent diagnosis of diphtheria or other bacterial disease without bacteriological aid, but it is just as true that no one who trusts empirically and implicitly to that aid can succeed.

In concluding what is evidently a very condensed and inadequate outline of the subject, I may summarize the points I wish to bring out thus: —

The problems of the public health laboratory, in relation to disease, are essentially epidemiological and preventive.

While primarily of the nature of applied science, they call for investigations the scope of which is very wide and involves peculiarly the study of the science of bacteriology itself as well as of the art.

ARTICLE XXXVII.

# SOME CASES OF CANCER TREATED BY THE X-RAYS.

By FRANCIS H. WILLIAMS, M.D.
OF BOSTON.

READ JUNE 12, 1901.

# SOME CASES OF CANCER TREATED BY THE X-RAYS.*

WHATEVER of value there may be to our community in what I am about to bring before you, is in a great measure due to the opportunities which a large hospital affords, and to the cordial interest which all the members of the staff of the Boston City Hospital have taken in this new therapeutic agent; to Drs. Burrell, Cheever, Cushing, Gavin, Lund, Monks, Mallory, Mason, Wadsworth and Bartol I am especially indebted.

Five years ago I began the study of the medical uses of the X-rays because I hoped they would be of value in the diagnosis of diseases of the chest; this hope has been more than fulfilled, and still another use for them has developed, namely, their employment as a therapeutic agent, of which no one at that time dreamed.

As there are several other papers to be presented to you, I will very briefly direct the attention of the Society to the treatment of external forms of cancer by this method, which is painless and harmless; and which thus far I have found to cause healing or improvement (this latter refers to cases still under treatment) in every case in which I have persistently carried it out; internal forms I will not discuss in this paper.

* Photographs of a number of patients, taken before treatment and after treatment, were shown. Patients who had had epithelioma, rodent ulcer and plasmona where the diseased area was healed over, were also presented at the meeting.

The exposure of a diseased area to the X-rays for a few minutes two or three times a week, for several weeks, is in general what is required. Where the growth is more extensive, a longer time is necessary; on the other hand, in early cases, perhaps ten exposures—or even less may suffice.

It will naturally be said that to carry this treatment to a successful issue in cases where the disease has spread over a considerable area, takes a long time; but patients are grateful if they find that a growth which they have seen slowly spreading for years—in some cases for fifteen or twenty years—begins to show signs of healing within two or three weeks; or to find that a rapidly growing epithelioma is arrested and then has diminished in size. Moreover, the method is a new one, and I can now reach results more rapidly and with less risk to the patient than I could at the beginning, and with further experience it will probably be possible to get results still more rapidly.

Another criticism that will be made on the use of this method is that the growth may return. It is yet too early to answer this. But if in any patient the disease should show a tendency to recur, it would probably be a comparatively simple matter to subdue it again.

The cases of cancer that I have treated include not only the smaller and lesser forms—growths in early stages of development in many cases—but also some which are inoperable. For example, a cancer of the eyelid in an old man. Operation in this case would mean the removal of the lid; and a plastic operation might be difficult or of doubtful result in an aged patient. (See Figs. 1 and 2.) Or, as another example, take a growth beginning near the eye which is now of thirty years' standing, on which two operations have been done—one to take out the eye. Finally, with the growth still extending, the wise and experienced surgeon of this patient decided that further operation was impossible. There was nothing to be done unless,

perchance, this new ray could be of service! Think of building any hope, even in so desperate a situation, on an invisible and little-tried ray, that exerts its action without pain and where to obtain a successful result the only duty of the patient is to sit still near the vacuum tube for a few minutes two or three times a week! This statement sounds incredible, and I should not ask you to have faith in it had not the rays given relief under the conditions outlined; for the diseased area on this patient is already nearly healed over.

Figures 3, 4, 5, 6, 7 and 8 are also illustrative of this method of treatment. The cuts are taken from my book on the Röntgen Rays in Medicine and Surgery, by permission of The Macmillan Company.

In justice to the method, I ought to state that some of the most striking cases are patients who have been treated in my office, but who live in other cities and who cannot be present at this meeting, and of whom I have no photographs.

In all the cases of which I have given photographs the diagnosis is based on a microscopic examination that was made in the pathological laboratory of the Boston City Hospital, by Dr. F. B. Mallory.

The microscopic examination has been used not only to make a diagnosis, but also in one case to observe what was going on in the growth during treatment. In this case a second specimen from a typical, rapidly growing epidermoid cancer was examined by Drs. Councilman and Mallory after treatment had been continued for some time. This examination showed that the central portion of the ulcerated area was entirely free from carcinoma, but, and this is an instructive point, that near the edges of the ulceration there were still a few small islands of epithelial cells. The persistence of these cells may have been due to the fact that the part of the hand where they occurred was more or less protected from the X-rays by the lead shield

which was used to prevent the rays from striking the healthy skin near the cancer, and this persistence shows that an area of apparently healthy tissue in the immediate neighborhood of the growth, as well as the growth itself, should be exposed to the action of the X-rays. It is not improbable that in some of the early cases recurrence will take place because this area was not treated.

In making X-ray examinations of the heart and lungs there is no risk whatsoever of a burn, but in treating external cancer there is such a risk, if care is not taken, because the tube must be much nearer the patient than in the former case. But I do not mean to suggest that such an accident cannot be avoided—quite the contrary—for all my cases have been treated without its occurrence.

In view of the great interest which is now taken in the cause of cancer, and the importance of knowing this cause, it will be instructive to consider for a moment what light my cases may throw on the nature of this disease. Some of these growths were typical epitheliomas; others were of the rodent ulcer type; still others were ulcers that were indurated and had persisted for months, or in some cases for years; clinically, these latter growths were epitheliomas, but when examined under the microscope they did not present the appearance typical of epithelioma, and were reported by Dr. Mallory as chronic, inflammatory tissue, or plasmona.

It is interesting to note that all these different classes of cases yielded to treatment by the X-rays. It may be that they were all due to one cause, and that these growths do not take on the appearances which we recognize under the microscope as cancer, for a longer or shorter period; or, on the contrary, they may be due to several different causes. In some cases these growths increased slowly; in others rapidly.

The healing effect which the radiation from an excited vacuum tube produces in a disease where the cause is

known, as in tuberculosis of the skin (lupus), taken in connection with the fact that syphilitic disease of the skin is said not to yield to the treatment; and further, in connection with the clinical picture which these healing cancers present, and that response to treatment in certain cases is immediate—sometimes within two or three days,—and also that in some cases the improvement continues after the exposures to the tube have stopped; all these facts, together with the microscopic appearances during treatment, suggest that cancer is probably produced by some living cause, as I have stated in a previous article, and that the X-rays interfere with its life.

In what light should we regard beginning cases? From the surgical standpoint operation would probably be advised, but I think we have now got far enough to justify the use of the X-rays in the early stages also, and to teach the community that these growths may be healed by a harmless and painless method, and thus few of them will be allowed to advance to serious dimensions through delay from fear of the knife. The first effects of the X-rays are apparent within two or three weeks.

Last evening Dr. W. F. Whitney, in the Shattuck lecture, made it clear that cancer is on the increase, not only in our State but in other parts of the world; and therefore I wish the more to bring before the profession, and through it as widely as possible to the laity, that we have in the X-rays a painless and a good method (how good it may take two or three years to decide) of treating many, if not all forms of external cancer. The first steps have already been taken, and now it only remains to determine the limits of this method and the best way of carrying it out; this is simply a matter of time and of careful study.

## DISCUSSION.

Dr. D. W. CHEEVER, of Boston: While reasonably sceptical as to new cures, facts compel me to yield assent to the occult power of the Röntgen ray in exciting a healing process in rodent ulcer, and in epidermoid cancer. I may add lupus, but of this I know only by hearsay.

A case of rodent ulcer, twice operated on, and now inoperable, is healing after twenty-two exposures to the X-ray, in the hands of Dr. Williams. These are facts. Whoever has, and who has not, seen in his native town the poor, blind victim of rodent ulcer, living for years, hopeless and an object of aversion, will rejoice in this prospect of relief!

Dr. W. T. COUNCILMAN, of Boston: The specimen which was removed from the tumor before treatment was begun showed that it was a typical epidermoid carcinoma. There were masses of epithelial cells growing in a connective tissue stroma which was rich in cells. That the tumor was growing rapidly was shown by numerous nuclear figures, indicative of cell division. The second specimen embraced the tumor and the adjoining healthy tissue. This showed that the masses of epithelial cells so prominent in the first specimen were necrotic and were included in the tissue as foreign bodies. Only at the edge of the specimen and deep in the tissue were a few small masses of living epithelial cells. The stroma had also undergone a marked alteration, the cells in it were not so numerous and it had the character of cicatricial tissue. What made this all the more remarkable was the presence in the tissue, in which the epithelial tumor cells were destroyed, of a number of sweat glands which were unaltered. A further interesting condition was a well marked endarteritis in the treated specimen, which had produced almost complete occlusion of the affected vessels. The exact way in which the treatment brought about the result must be the subject of further investigation. It seemed to exert a destructive action on the foreign cells in the tissue not affecting the normal elements. What part the endarteritis played in the process is uncertain.

FIG. 1.—EPIDERMOID CANCER OF EYELID, BEFORE TREATMENT BY THE X-RAYS.

FIG. 2.—EPIDERMOID CANCER OF EYELID, AFTER TREATMENT BY THE X-RAYS.

FIG. 3.—EPITHELIOMA OF LIP, BEFORE TREATMENT BY THE X-RAYS.

FIG. 4.—EPITHELIOMA OF LIP, BEFORE TREATMENT BY THE X-RAYS.

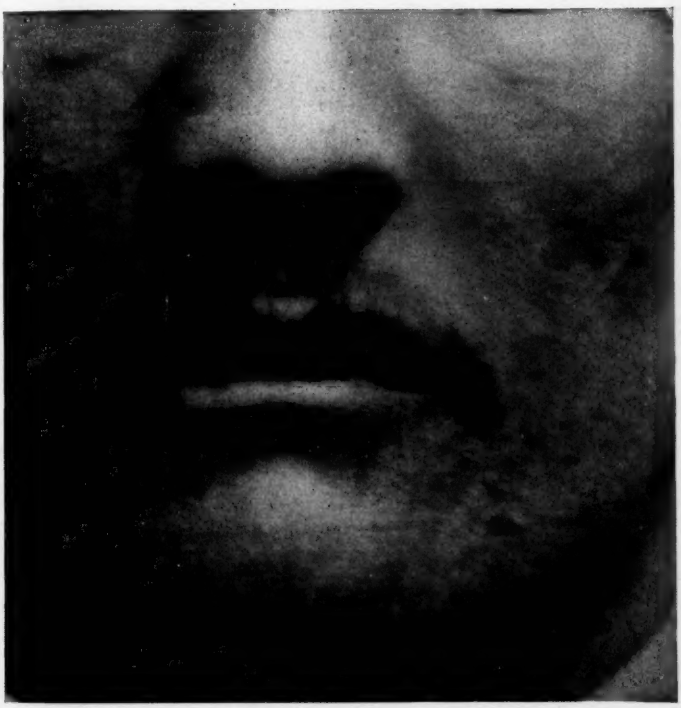

FIG. 5.—EPITHELIOMA OF LIP, AFTER TREATMENT BY THE X-RAYS.

FIG. 6.—EPITHELIOMA OF LIP, AFTER TREATMENT BY THE X-RAYS.

FIG. 7.—RODENT ULCER, BEFORE TREATMENT BY THE X-RAYS.

FIG. 8.—RODENT ULCER, AFTER TREATMENT BY THE X-RAYS.

ARTICLE XXXVIII.

# INTRACRANIAL PRESSURE AFTER HEAD INJURIES.

By WALTER B. CANNON, M.D.,
OF BOSTON.

READ, BY INVITATION, JUNE 12, 1901.

# INTRACRANIAL PRESSURE AFTER HEAD INJURIES.

## Introductory Remarks,
## By William N. Bullard, M.D., of Boston.

The experiments about to be described by Dr. Cannon were instituted by him in the Physiological Laboratory of the Harvard Medical School at my suggestion, for the purpose of throwing some light, if possible, on the cause of certain phenomena which had been observed clinically and to which I had previously frequently called the attention of the profession. The object of this work was (1) to attempt to prove experimentally the fact of the increase of brain pressure after trauma; (2) to determine the cause of such increase, if it existed.

The first object has, I feel convinced, been attained. How far the second has been accomplished I must leave to others to determine.

For these very valuable experiments Dr. Cannon is entirely responsible, both as regards methods and conclusions, and deserves all the credit therefor.

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The clinical phenomena attending traumatic injuries to the brain have been made prominent in this country by the studies of Dr. W. N. Bullard and Dr. G. L. Walton. In cases of brain lesion there is usually a history of a blow or a fall on the head, or evidence of the rupture of an intracranial blood vessel. The most notable and constant

of the primary symptoms is unconsciousness, which may persist until death or may pass away. If consciousness returns, it is commonly lost again, often gradually increasing dullness passing into stupor. The final appearances, whether consciousness is recovered or not, are deeper and deeper stupor, then coma, from which the patient cannot be aroused, and ultimately death. In this final stage certain typical signs manifest themselves. The temperature characteristically rises as the time of death approaches, the pupils do not react to light, there is stertorous breathing and slow heart beat. Sometimes there is paralysis of the face and limbs, though there may be clonic spasms in various muscles. Passage of urine and feces is also an occasional occurrence.

Between these symptoms and the symptoms produced in animals upon increasing intracranial pressure, there is a striking similarity. The resemblance between clinical symptoms and experimental phenomena has led to the application of the term "pressure symptoms" to the peculiar symptom complex following brain injury, and the evidence of intracranial pressure observed in these cases on operation confirms the validity of the term.

From a study of the pathological alterations after traumatic lesions of the brain it is clear that there may be hemorrhages and lacerations so severe as to result in almost immediate death. It is also clear that in many instances of fatal issue the initial lesions—the scattered minute thrombi and punctate extravasations always present in contusion—are so slight as to be in themselves no adequate cause of death. That this inference is true is indicated by the recovery of consciousness after the initial loss of it—a result hardly to be expected if the primary lesions were intrinsically fatal. But not infrequently in these cases the recovery of consciousness is followed by a slow subsidence of conscious life, with a progressive increase of the so-called

pressure symptoms until death supervenes. Leyden long ago showed that, in order to produce death, intracranial pressure must equal arterial pressure. Apparently what occurs in these puzzling cases is this: The intracranial pressure somehow rises higher and higher until it equals blood pressure in the arteries. At this point the circulation in the cerebral blood vessels comes to a standstill, the vital centres in the medulla no longer receive their normal nutrition, they become paralyzed, and life ceases. Now the question presents itself, In what way is the intracranial pressure gradually and progressively increased until the pressure in the blood channels is overcome and death results? It is clear that the pressure symptoms which slowly manifest themselves are dependent on secondary processes following the primary lesions of the brain. It is certain, also, that *œdema* of the tissue follows the primary lesions of the brain. Is there any causal relation between these two phenomena?

The classic theory of Bergmann, put forth to explain the secondary increase of cerebral pressure, starts with the assumption of an intracranial hemorrhage, which acts as a foreign body within the cranium, causing an obliteration of the capillaries and veins in the region it occupies. The result is stasis of blood in the obliterated area, which results in high blood pressure in the border areas. This high blood pressure leads to an increased transudation of fluid, because plasma may pass, according to the theory, more easily into the brain substance than blood through the compressed capillaries. The transudation will increase the volume of the foreign body and compress other capillary areas, and so the process runs till death occurs.

It should be noted that by this theory it is assumed that transudation occurs because of high blood pressure in the capillaries of the border areas. This is due, however, not

to a general increase in blood pressure, but to external pressure in the brain substance about the vessels, increasing until it partially overcomes the internal pressure in the capillaries themselves. Since there is this higher pressure *outside* the vessels, transudation from them must be well-nigh impossible.

But even the assumption that plasma passes from the vessels does not remove every difficulty. For if the secondary increase in intracranial pressure is a result of transudation, this pressure must be dependent on the pressure of the plasma. The pressure of the plasma is in turn dependent on blood pressure and is as much *less* than blood pressure as the resistance which the tissue about the capillaries offers to the outflow from them. The difficulty now arises in attempting to induce a method of compression, manifestly less effective than arterial pressure, to overwhelm arterial pressure. In the end, by this reasoning, the direct force of the blood through the free ways of the vessels must be greater than the lessened pressure of the transudate, and the flow will persist.

This and other theories, moreover, have the grave defect of not regarding the processes taking place in the portion of the brain substance in which the circulation is impaired. It will be shown that in this neglected region swelling and pressure occur wholly independently of any blood pressure whatever.

About two years ago Dr. W. N. Bullard pointed out to me the importance of knowing the cause of increased cerebral tension following injuries to the head, and since that time I have busied myself at intervals with the problem.

My experiments show that at the moment of injury the intracranial pressure rises to a height sufficient to check the blood-flow into the brain. Immediately after the injury the general blood pressure usually rises for a moment, then falls. Thereafter a gradual recovery of normal blood pres-

sure occurs, with a simultaneous increase in the extent of the pulsation of the brain.

Occasionally after injury to the head there is paralysis of the respiratory centre and cessation of respiration. If artificial respiration is persisted in and the heart action remains strong, the centre will resume its function—an observation of practical importance, confirmatory of Horsley, Polis and Kramer.

The normal cerebral pressure is about 13 cm. of water, and after injury the brain pressure may rise to an average of 25 cm. of water. Men retain consciousness, however, in the midst of the paroxysms of strychnine poisoning, when, according to Hill, the brain pressure must equal at least 68 cm. of water. The primary loss of consciousness after a blow on the head is apparently due to the circulatory disturbances, though minute changes in nerve cells must also be considered. But the increase of brain pressure to 25 cm. of water is clearly not sufficient to account for the pressure symptoms observed in clinical cases. There must be other secondary processes at work to produce such results.

Before enquiring into these secondary processes, three conditions must be regarded: the anatomical arrangement of the cerebral blood vessels, the changes caused by injury, and the effect of these changes on the circulation in the brain.

On the surface of the brain the vessels form a vast canaliculate reservoir with free communication from one part to another. From this common reservoir there pass into the brain substance the nourishing arteries; some to the cortex, some to the deeper fibre tracts. Each of these vessels is independent, terminal, not anastomosing with other vessels from the surface or with branches from the central supply. Interruption of the arterial supply to a part does not therefore permit the easy establishment of a collateral circulation.

The pathological changes in contusion of the brain, which exists in all cases of intracranial injury, are a diffuse formation of thrombi, punctate extravasations and thin patches of hemorrhage in the meninges. Owing to the terminal nature of the arteries the general formation of thrombi and extravasations must result in a general diminution of the blood supply of the injured tissue. Whether the interruption of the flow causes stasis or anæmia, the result must be impaired nutrition of the brain. The problem of the secondary increase of intracranial pressure is essentially the determination of the behavior of brain tissue deprived of its normal nutrition.

From studies of such widely different structures as unicellular organisms, the muscular tissue of the frog, and nerve cells, it has been proved that lack of oxygen causes protoplasm to absorb water. Loeb ligatured the leg on one side in a large number of frogs and after a time removed and weighed the two gastrocnemii muscles. Normally the two muscles have the same weight, but under the circumstances of the experiment the muscle deprived of its blood supply took up water so that in forty-eight hours it had gained 15 per cent. in weight. Loeb concluded that his experiments left no doubt that the assumption of water by a muscle deprived of its blood supply is due to chemical changes in the muscle increasing the internal osmotic pressure, and that these chemical changes are probably due to lack of oxygen.

As has been noted, the initial changes in the injured brain are hemorrhage and contusion, with thrombi and extravasations. The result of these changes is an impaired blood supply to the injured region and a consequent lack of oxygen. Both experiment and clinical observation prove that œdema of the tissue follows. Bergmann says: "One finds about the region of injury merely a small zone which

is scattered with specks of blood and colored yellowish red; farther on, as far as the swelling reaches, the brain substance appears moist, glistening, soft,—that is, highly œdematous." Now the question arises, Is this œdema, this result of primary pathological changes, this concomitant of the secondary pressure symptoms, a passive transudate due to blood pressure, as has been held hitherto; or is it the effect of chemical changes in the brain substance, resulting from diminished blood supply and causing the taking of water into the tissues by increased osmotic pressure? The difficulties encountered by the former process when required to produce the equivalent of arterial pressure have been noted; the latter process provides a force amply sufficient to overcome any possible blood pressure.

Normal salt solution has the same osmotic pressure as the blood, and for this reason may be injected into the vessels without injury. Animals have been infused with the solution even to 110 per cent. of body weight within 143 minutes without producing any general œdema. Since normal salt solution is isotonic with the tissues, so that in its presence water does not pass into them when they are nourished, the passage of water from the solution into the tissues when they are not nourished indicates an increase of osmotic pressure within them. Upon this statement was based the following series of experiments with brain tissue.

Normal salt solution was allowed to run through the cerebral arteries of a cat, under a pressure equal to normal blood pressure. The brain very soon began to take up water, and the pressure of the brain against the skull, measured by a mercury manometer, began to increase. In half an hour the flow through the vessels had markedly diminished and the cranial pressure had increased to three times the normal amount. In four hours and a half the pressure of the brain nearly equalled blood pressure and the flow through the vessels had practically ceased—a few

hours later the brain had taken up water until it completely filled the cranial cavity.

Is this increase of pressure, this taking up of water, due to transudation, or is it the result of an active process in the tissues themselves? That brain tissue, deprived of its proper blood supply, will take up water from a solution isotonic with the blood, entirely independently of any mechanical pressure of the solution, can readily be demonstrated by removing a brain and placing it in 0.8 per cent. sodium chloride solution. Under these circumstances it will almost immediately begin to increase in weight, during the first four hours most rapidly, and thenceforth slowly and persistently for days. In one instance, after five days the brain had increased one-third in weight and in size. Since a diminution of the intracranial space by one-twelfth causes death, it is evident that impairment of the nutrition of the brain as a whole may cause it to take up water to a degree far greater than that necessary to produce death. Now what is true of the brain as a whole may be assumed to be true of any parts deprived of their blood supply, and localized regions thus affected would take up the water of the plasma from neighboring regions and swell in the same manner in which the brain as a whole will swell. Indeed, histological observation of injured brain tissue has shown that the dendrites and bodies of the nerve cells swell as they degenerate and vacuoles from within them precisely as in unicellular organisms deprived of oxygen.

Observations on brain and nerve cells prove that blood pressure is not a necessary factor in causing cerebral swelling and œdema. It may be that there is transudation from the cerebral blood vessels when intracranial pressure is locally increased, but it has not been shown that the transudation is a mechanical result of the increased pressure, nor that, even with a mechanically produced transudate, the pressure resulting therefrom is sufficient to cause death.

Moreover, the factors increasing the osmotic pressure of the tissues have not been regarded. Hill states: "Under normal pressures the secretion and absorption of cerebro-spinal fluid does no doubt follow osmotic laws." He overlooks the fact that deprivation of nutrition causes chemical changes within tissues, which affect osmotic pressure, a force far beyond mechanical blood pressure in its powers.

A conception of the force involved in the increase of weight when the brain is placed in salt solution, may be obtained by means of a method used by Loeb. A brain was placed in a 2.0 per cent. solution of sodium chloride. During the first four hours there was a diminution of weight because the osmotic pressure outside was greater than that within the tissues. Soon there was a sharp rise, however, and after about ten hours the slow, persistent increase in weight began. The osmotic pressure of a 2.0 per cent. sodium chloride solution is about 14.5 atmosphere. The pressure within the tissues must have developed to this great height in order that the water should pass into them. Inasmuch as the osmotic pressure of an 0.8 per cent. salt solution is only 5 atmospheres, there is evidence of a pressure increase of 9 atmospheres, which is about 57 times ordinary blood pressure. Naturally such a pressure never becomes fully operative in the tissues—disruption must take place long before that result could occur. The internal osmotic pressure is nearly balanced by the external, but because of the greater internal pressure fluids pass inwards till the mechanical pushing of the tissues overcomes the push of the blood, thereby shutting out the source of fluid supply. It is clear, therefore, that in the chemical changes taking place in dying tissues there is a force present abundantly able to overwhelm the blood pressure.

The mischief arises because the brain is surrounded by a rigid case. Swelling of a part consequently compresses the only compressible portion of the cranial contents,—the

blood vessels. Thereby new areas are shut out from normal blood supply, and changes now take place in these tissues as well, with the result that water passes into them; thus the swelling spreads until the blood flow is so greatly excluded from the brain that life is no longer possible.

It is thus, I believe, that the cases of head injury resulting in death from secondary increase of brain pressure would be explained. Injuries to the brain interfere with its proper blood supply. Such interference causes an increased osmotic pressure within the tissues and a consequent taking up of water from the surrounding plasma. The swelling and œdema of the brain after head injuries, therefore, is not due to passive transudation, as Bergmann and others have maintained, but is the result of an active process in the tissues themselves, a force many times greater than blood pressure, and amply sufficient to produce all the pressure symptoms and account for all the signs of intracranial tension which the clinical cases of cerebral trauma often manifest.

ARTICLE XXXIX.

# THE ORIGIN OF OXALIC ACID FROM PROTEIN AND PROTEIN DERIVATIVES.

By ARTHUR E. AUSTIN, M.D.,
OF BOSTON.

READ, BY INVITATION, JUNE 12, 1901.

# THE ORIGIN OF OXALIC ACID FROM PROTEIN AND PROTEIN DERIVATIVES.

THE question as to whether Oxalic Acid is a metabolic product or is only due to the elimination of that taken in the food when found in urine and feces, has become more and more a matter of importance during the last few years. Helen Baldwin, in an excellent article in the Journal of Experimental Medicine (volume 5, number 1), has given a very valuable reference to the investigations made by others, and has also apparently established the origin of oxalic acid from the fermentation of carbohydrates without having been able to establish what fungus or organism appears to be the originator of this product. Nevertheless, this effort of hers is a step in the right direction, and has offered me a great deal of assistance in the attempt which I have made in elucidation of this problem. In addition to the references given by her there have also appeared the investigations of H. Lūthje, Zeitschr für klin. Medic., 35–271–282, from the abstract of which, in the Jahresbericht für Thier-Chemie (Vol. 28—584), we learn that in metabolic examinations on dogs, he has been unable to show any connection between the amount of oxalic acid eliminated during fasting and during feeding upon large amounts of sugar, fat and horse flesh; nor was the amount of oxalic acid eliminated from a diet of thyroid gland and nuclein distinctly increased.

During the last year investigations have been made by Salkowski (Berlin. klin. Wochr., 37–20), who has demonstrated upon dogs no particular increase in the elimination of oxalic acid, in mixed diet over that in hunger, or from feeding upon eukasin, which is a preparation of casein. Further, F. Lommel (Centralbl. f. d. medic. Wissensch., 1899, number 16), has demonstrated that, while the greater part of the oxalic acid is the result of metabolic processes, a diet, rich in albumins, gave no special increase in the elimination of oxalic acid. He also concludes that the oxalic acid in the urine and feces forms only a small part of that which is ingested; in other words, a great portion of it is decomposed in the body. He found that food rich in nuclein gives an increase in the amount of both oxalic and uric acid eliminated. Encouraged by these results to follow up this subject a little more closely, experiments were made with digestions of different forms of albuminous materials, excluding the fermentative processes by means of thymol solution. Parallel experiments were also made with similar solutions of albuminous material, encouraging fermentation as much as possible both by excluding the thymol and by infecting the solutions with fecal matter. This was done, notwithstanding the fact that Salkowski and Leube, in their work on the examination of urine, state distinctly that oxalic acid has never been produced from albuminous material by means of digestions. In this connection efforts were also made by means of heating albuminous material with barium hydrate at a temperature of 150 degrees for periods of eight hours and over, to produce oxalic acid from the same. The detailed results of these experiments appear as follows:

(1) 1 gram of casein and ½ gram barium hydrate were heated in a closed, sealed tube for 8 hours, then the substance was extracted with boiling alcohol, acidified with HCl, three times, the alcoholic extracts united, filtered, eva-

porated to dryness, taken up with distilled water, decolorized with animal charcoal, filtered, precipitated with $CaCl_2$, then neutralized with ammonia and weakly acidified with acetic acid. This was allowed to stand for several days, but no oxalate of calcium was found.

(2) The same experiment was performed with 5 grams casein, 5 grams peptone, 5 grams gelatine, 5 grams egg albumin, but always with negative results.

(3) 1 gram of peptone, 1 gram albumin, 0.2 grain pepsin, 50 mgs. HCl, were digested in a brood oven two days, then removed and subjected to the previous process for the extraction of oxalic acid, but none could be found. A control test was made with the same ingredients, except the pepsin, which was inoculated with a small portion of feces. The extraction was made as before, but again with negative results.

(4) 5 grams of egg albumin, 0.2 gram pancreatin, 100 c.c. 0.3% solution of sodium carbonate were digested for a week and, as a control, the same solution less the pancreatin, infected with feces, was digested for the same period. Both were extracted as before, but no oxalic acid was found.

(5) 6 grams boiled white of egg, 800 c.c 0.3% sodium carbonate solution and 1 gram pepsin were digested for ten days and a similar control with the same ingredients without pepsin, but infected with feces, was digested for the same period, and then again extracted, but also with negative results.

(6) 10 grams casein, 1 gram pancreatin, 200 c.c. 0.3% sodium carbonate solution were digested for two days and controlled with similar ingredients less the pancreatin, infected with feces, which were digested for the same period; then both were extracted, but again with negative results.

(7) 10 grams of gelatine, 1 gram pancreatin, 500 c.c. 0.3% sodium carbonate solution were digested for a period

of 6 days and controlled with the same ingredients less pancreatin, infected with feces, digested for the same period when both were extracted. It was again impossible to find any evidences of oxalic acid in the extract. The impossibility of obtaining oxalic acid from gelatine seems especially surprising, since this substance from its molecule containing a large carbohydrate group would seem to explain the successful efforts made in obtaining this substance from sugars. Perhaps the proper organism was not introduced, and, until that is determined, we can never be sure that oxalic will or will not be formed.

It was evident that all these substances had undergone thorough decomposition, as proved by the presence of ammonia where barium hydrate was used, of peptones in the digestive products, and indol in most of those which had undergone bacterial decomposition; yet no oxalic acid was found in any. It is possible that the method was too crude for the detection of such small quantities, yet it is one recommended in toxicological examinations. The method of Bischoff was also employed in some of these examinations, in order to exclude the possibility of calcium carbonate being present, which was frequently found and which simulates the oxalate of calcium very closely in its oval forms. This method consists in dissolving the colored residue produced by the first precipitation with calcium chloride and ammonia, which fails to dissolve by the addition of small quantities of acetic acid. This can be readily accomplished by sedimenting this residue with the centrifuge and then digesting it in 30% acetic acid after washing two or three times with water. The remnant is then thrown upon a filter, washed once more with water and then dissolved upon the filter with hot 25% HCl, and after cooling the filtrate is again neutralized with ammonia. Even in cases where this method was employed the results were invariably negative.

(8) The next attempt was made with a much more complex substance—brain matter—and 100 grams of evenly divided calves' brain were digested with 500 c.c. .3% sodium carbonate solution, .5 gram pancreatin, with a drop or two of thymol for five days. It was then extracted in the above mentioned manner with the Bischoff modification and positive results were reached; both oval and quadrate prisms with four-sided pyramidal ends were found and proved to be oxalate of calcium crystals, both by their insolubility in acetic acid as well as by the absence of carbonic acid which excludes calcium carbonate.

As a control test, 100 grams of calves' brain, 500 c.c. .3% sodium carbonate, .5 grams pancreatin, inoculated with fresh feces, were digested in a brood oven for 5 days and then extracted in the usual manner. Again, oxalate of calcium crystals were found, both oval and octrahedral, which were verified in the above manner. Of course there remains only the possibility that the oxalic acid was preformed in the brain substance, since Salkowski found in one kilo of calves' liver respectively 10.66 and 8.73 mgms. of oxalic acid, and in a kilo of ox liver 12 mgms. oxalic acid, less in muscles and none in pancreas. To prove this possibility one kilo of pigs' brain finely chopped was cooked out repeatedly with water with the addition of enough acetic acid to coagulate the albumin, and the concentrated watery extracts were manipulated according to the newer method of Salkowski, which is as follows:

200 c.c. of the fluid to which 20 c.c. of hydrochloric acid, specific gravity 1.12, have been added, is shaken three consecutive times with ether-alcohol 1 to 10 and then the alcoholic-ethereal extracts evaporated to a small volume, 15 to 20 c.c. of water added, when it is again evaporated with the addition of water, if needed, to render it clear, reduced to 20 c.c., whereupon it is allowed to cool and filtered clear. The precipitate is washed lightly with water and the

filtrate neutralized lightly with ammonia, the acetic acid added until it is weakly acid, and then 1 to 2 c.c. of 10% calcium chloride added. By this method, which appears very satisfactory with Salkowski, there was obtained oxalic acid from the fresh well-washed brain substance. In the second attempt, in which a quantitative estimation was made, 8.51 mgms. of oxalic were found in one kilo of the brain matter. Since this substance is so almost universally found and in such varied tissues, it is very probable, perhaps almost demonstrated, that it is a metabolic product. This is further substantiated by the fact, as demonstrated by Salkowski and others, that even after 14 days of absolute fasting this acid can still be found in the urine, and furthermore that on account of the insolubility of the calcium salt, the form that the acid must assume after entering the intestine, absorption must be very difficult, apart from the stomach, while the short stay of the food in this organ still further reduces the possibility of its absorption. Assuming, then, that it is a metabolic product and can be found, as in this instance, in the brain, we have to consider some of the various substances of which the brain is composed, by means of the retrograde metamorphosis of which it is possible that oxalic acid may be derived. Some of these which were examined were lecithin, glycocoll, nuclein, mucin, stearin, tyrosin and uric acid. There is also a group of protagons which I hope at no distant day to investigate, and which form a specially hopeful field on account of the carbohydrate group which their molecule contains, and which is so marked that already the estimation of protogon in nerve tissue has been made by the strength of their reducing power when converted to sugar by cooking with acid. A detailed account of these experiments is given briefly as follows:

(10) 50 mgms. of glycocoll and 200 mgms. of ammonium carbonate were heated in a closed tube at 150 degrees for

8 hours. The resulting product was digested for 24 hours with water and enough hydrochloric acid to make it slightly acid to litmus, then filtered, washed, neutralized slightly with acetic acid, a small quantity of calcium chloride solution added to ensure an excess of calcium, and then half the quantity of alcohol added; a slight modification of the method of Dunlop, but no crystals of oxalate of calcium were found.

(11) 500 mgms. of stearic acid, 1 gram of ammonium carbonate were heated in a closed tube at 150 degrees for 8 hours and the product extracted in the same way, but no oxalic acid was found.

(12) 100 mgms. tyrosin and 1 gram barium hydrate were heated in a closed tube 8 hours at 150 degrees and extracted in the same way, but no oxalic acid was found.

(13) 100 mgms. nuclein and one gram barium hydrate were heated and extracted in the same way. Crystals were found, both round and oval, which were evidently calcium carbonate, since they were soluble in acetic acid with effervescence.

(14) 100 mgms. lecithin and 1 gram barium hydrate were heated and extracted in the same way. No oxalate crystals were found, but crystalline needles, which were probably stearic acid.

(15) 100 mgms. uric acid and 1 gram barium hydrate were heated and extracted and numerous well-formed crystals of calcium oxalate were found.

(16) 100 mgms. mucin from bile were treated in the same manner, but with negative results. As is readily seen, all these results were negative with the exception of uric acid (15), a result which shows the close association between uric and oxalic acids, probably through the intermediary product known as oxaluric acid and brought about by a combination of the processes of oxidation and hydration, as shown by the following formula:

$$C_5H_4N_4O_3 + O_2 + H_2O = C_3H_2N_2O_3 + CO(NH_2)^2 + CO_2$$

Uric Acid — Parabanic — Urea

$$C_3H_2N_2O_3 + H_2O = C_3H_4N_2O_4$$

Parabanic — Oxaluric acid

$$C_3H_4N_2O_4 + H_2O = CO(NH_2)_2 + C_2O_4H_2$$

Oxaluric — Urea — Oxalic acid

It is noticeable that during this reaction two molecules of urea are given off. As the process of hydration seemed most actively employed in the meagre results already obtained, all of these products were submitted to mixed fermentation in hopes that the activity of the ferment organisms might accomplish what other chemical means had failed to do, as follows:

(17) 100 grams each of lecithin, uric acid, glycocoll, mucin, nuclein, tyrosin and stearic acid were suspended in 50 c.c. water, each in its own flask without stopper, and placed in the brood oven at a temperature of 38–40 degrees for 23 days, when all were removed and examined under the microscope for any changes that may have occurred; no change was found in any except the nuclein, which showed numerous yeast cells, and the glycocoll which showed a mass of mycelium.

Each of these solutions was then alkalinized slightly with sodium carbonate and again placed in the oven for 21 days, when they were all extracted by the modified method of Dunlop. The nuclein, lecithin, mucin, tyrosin and stearic acid showed negative results, while the glycocoll and uric acid both showed beautiful crystals of calcium oxalate, whose origin was, of necessity, from these substances, and which were verified by their insolubility in acetic acid. These experiments, repeated with these two substances, showed the same results. If, then, this conversion of uric acid to oxalic could take place by bacterial decomposition in pure solution, it was to be anticipated that the same ac-

tion would take place in urine, where both were present, if allowed to decompose, and an explanation would be offered of the well known increase of the calcium oxalate crystals in old urine, an occurrence hitherto attributed to the conversion of acid phosphate, the natural solvent of this substance, to the neutral and alkaline phosphates which have not this power, and the consequent precipitation of the oxalate. This could explain a precipitation, but never an increase, while an increase would naturally explain a precipitation in a previously saturated solution. Furthermore, Huppert in his Harnanalyse, page 321, says that oxaluric acid may undoubtedly arise from the action of micro-organisms on uric acid, while oxalic acid may be obtained by splitting the oxaluric acid by boiling, but gives no evidence to substantiate the former statement. Here we have been able to carry this evolution still farther and to say that micro-organisms can also split off the oxalic acid without the aid of cooking. The series of experiments which follows was based upon the fact that if the uric acid and oxalic acid were estimated quantitatively in a urine while fresh, and then they were subsequently estimated in the same urine after it had undergone fermentation, the comparative amounts of these two substances found would verify or disprove this assumption; accordingly, from several liters of fresh well mixed urine, two portions of 500 c.c. each were taken for the oxalic acid estimation and two portions of 150 c.c. for the determination of the uric acid. For the former, the method of Salkowski, extraction by ether and calculation from the CaO formed was employed, and for the latter, the method of Woerner, precipitation by ammonium chloride and estimation of its nitrogen by Kjeldahl, from which the uric acid was calculated. The remainder of the urine was placed in the brood oven in an unstoppered bottle, and after a period of from five to seven days removed, cooled to temperature of the room, well

mixed, and again portions of the same amount taken for two more parallel determinations of each acid. The results of these experiments, six in number, are best shown in a table which follows:

| FRESH URINE. | | | | SAME URINE DECOMPOSED. | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mgms. | | Mgms. | Period. | | Mgms. | | Mgms. |
| Uric | 1–25.2<br>2–27.3 | Oxalic | 1–9.96<br>2–9.32 | 5 dys. | Uric | 1–10.29<br>2– 9.45 | Oxalic | 1–12.21<br>2–12.85 |
| Uric | 1–45.36<br>2–45.36 | Oxalic | 1–3.21<br>2–3.53 | 4 " | Uric | 1–32.76<br>2–32.76 | Oxalic | 1– 8.99<br>2– 8.99 |
| Uric | 1–69.09<br>2–61.3 | Oxalic | 1–8.67<br>2–8.67 | 4 " | Uric | 1–64.28<br>2–69.3 | Oxalic | 1–13.17<br>2–Lost |
| Uric | 1–61.47<br>2–Lost | Oxalic | 1–2.88<br>2–3.21 | 5 " | Uric | 1– 4.2<br>2– 6.3 | Oxalic | 1– 5.14<br>2–Lost |
| Uric | 1–58.38<br>2–67.2 | Oxalic | 1–5.46<br>2–Lost | 2 " | Uric | 1–41.48<br>2–Lost | Oxalic | 1– 9.64<br>2–10.21 |
| Uric | –29.4 | Oxalic | –9.64 | 5 " | Uric | –25.2 | Oxalic | –12.05 |

Considering the average amounts of uric acid, we find a diminution respectively of from 26.25 to 9.87 mgms.; from 45.36 to 32.76 mgms.; from 61.47 to 5.25 mgms.; from 62.79 to 41.48, and from 29.4 to 25.2 mgms. In one case only was there no diminution. The oxalic was increased on averaging parallel results from 9.6 to 12.05 mgms.; from 9.64 to 12.53 mgms.; from 3.37 to 8.99 mgms.; from 8.67 to 13.17 mgms.; from 3.04 to 5.14 mgms., and from 5.46 to 9.925 mgms. in the same urine. No constant ratio can be discovered between the increase in oxalic and the loss in uric acid, but since, if this transformation takes place according to the formula given above, two molecules of urea result as side products, it is easy to see the difficulty arising from a close adherence to the formulary demand in increase. A more desirable way would be to estimate the increase in urea, were it not that in a decomposing urine the urea is also converted into ammonium

carbonate as soon as formed, with loss of the ammonia by evaporation.

For instance, in the first experiment the formulary demand for oxalic acid from a loss of 16.38 mgms. uric acid would be 8.77 mgms. oxalic; that is, multiplied by the factor .5357, if all were converted to the latter, while we actually find but 2.9 mgms.; but when we remember that parabanic and oxaluric acids are formed as intermediate products, and, though both present, resist the method of extraction used for oxalic acid and thereby escape estimation, we can easily see why no constant relation between the loss of uric and the gain in oxalic acid can be expected. From the transformation of uric acid to oxalic, it is, perhaps, allowable to advance their common origin from the same substances, a supposition which has already found supporters in those who have found a common increase in diabetes mellitus, severe jaundice, etc., and also those like Lommel, who from experiments in metabolism have demonstrated that foods rich in nucleins produce a common increase in both these; nor can it be denied that this forms a still further demonstration of the fact that oxalic acid is a metabolic product. To return now to glycocoll, which was also found to be transformed, partially, at least, by microorganisms, into oxalic acid, we find that this has a close relation to gelatine, from which it was obtained by Braconnat, by the process of cooking with sulphuric acid, and by Nencki from decomposition in pancreatic digestion. Furthermore, Schultzen and Nencki, as well as Salkowski, found that partially, at least, glycocoll was converted to urea when fed to animals, and V. Knieriem to uric acid when fed to birds; but neither attempted to demonstrate what becomes of the remainder, though Lommel found an increase in the oxalic acid eliminated when animals are fed upon gelatine.

Karl Spito (Zeit. f. phys. Chemie, 28–185) has also

shown that the glycocoll is not confined to gelatine, but can be obtained from fibrin, hæmoglobin and globulin by cooking them with sulphuric acid in an autoclave (but he was not able to obtain it from casein), thus demonstrating that glycocoll is a much more common constituent of the albumen molecule than generally supposed. In order to clear up some of these obscurities, a female Newfoundland dog, weighing 16.1 kilos, was placed for six days upon a diet of dog bread of known weight and nitrogen content, catheterized three times a day, and each three days urine worked for oxalic acid by Salkowski's method for urea by the method of Mörner and Sjöqvist, and for total nitrogen by Kjeldahl, of which two parellel estimations were always made. The feces were also collected in three day periods, dried with the aid of alcohol, pulverized, passed through a fine sieve, weighed, two estimations of nitrogen made of the same, and the remainder extracted by 1% alcoholic solution of hydrochloric acid with the aid of a cooler, converted to a watery solution, and the oxalic acid estimated by the method of Salkowski. Then there followed, under the same conditions, six days' feeding with dog bread and increasing amounts of gelatine, and finally three days of dog bread with hypodermic injections of glycocoll; a small portion of every cake in every box, containing about 400 grams in groups of six boxes, was taken, the whole pulverized, and two portions of about one gram each of the mixed mass were employed for estimation of the nitrogen by Kjeldahl. The nitrogen of the gelatine was estimated, while the nitrogen of the glycocoll was calculated from the formula, since this was obtained from Merck, and such a course seemed justified. The results are best expressed in tabular form which follows, and then certain features will be discussed in their turn. The urine was placed on ice as soon as drawn, with a drop of thymol solution, and the dog was given water ad libitum. A day was allowed to elapse after

each period before the next was begun, and after gelatine and glycocoll periods, urine and feces were collected for twenty-four hours after last feeding or injection; 397 grams of dogs' bread, consisting of portions of all the cakes used, worked for oxalic acid, showed only a few crystals, too few to weigh. (Page 738.)

One very noticeable fact was that coincident with the gelatine and glycocoll feeding, there appeared in the urine a body which reduced both Fehling and Nylander solution slowly, was a left turner in the polariscope to the extent of .2 of 1% in a urine of 1072 and 1220 c.c. in amount, gave a Tollens reaction, was not fermentable, nor would it form an osazone. This body can be no other than paired glycuronic acid, for upon cooking this body with 1% sulphuric acid, it first became inactive in the polariscope and then a right turner. While not dextrose, still it must be allied, and as it occurred coincident with a decided increase in oxalic acid (32 to 43 and 70 mgms)., it must show some connection in these two metabolic anomalies, a fact long suspected but never absolutely proven. This condition is such as is seen in diabetes mellitus, where with great loss of sugar in the urine, there is usually increased oxalic acid eliminated. This can have been due only to the gelatine and glycocoll, for before and after these substances, no such body was found, but it persisted for forty-eight hours after the cessation of the glycocoll. It is also worth while to note the ratio of urea nitrogen to total nitrogen, which amounts in the first period, to 89.63%; in the second, 85.71%; third, 96.27%; fourth, 91.05%; fifth, 89.45%, since it demonstrates that neither glycuronic nor oxalic acid can pair to any great extent with ammonia, else the urea nitrogen in the latter periods would be diminished. The increase of oxalic acid, after feeding upon gelatine, cannot admit of a doubt, nor can the fact that this acid must be formed apart from the intestinal tract, since one would

| INGESTED. | | | | | | ELIMINATED. | | | |
|---|---|---|---|---|---|---|---|---|---|
| Bread | Total N. | Gelatine | Total N. | Glycocoll | Total N. | Urine | Total N. | Urea | Oxalic |
| 1095.37 | 24.319 | | | | | 820 | 11.594 | 22.238 | .01844 |
| | | | | | | | 5.22 | | .0016 |
| | | | | | | Feces | | | |
| | | | | | | 90 | 16.814 | | .02004 |
| 1572.53 | 45.105 | | | | | Urine | 11.627 | 21.347 | .03119 |
| | | | | | | 565 | 5.22 | | .0016 |
| | | | | | | Feces | | | |
| | | | | | | 90 | 16.847 | | .03279 |
| 1279 | 38.945 | 120 | 7.644 | | | Urine | 24.162 | 49.826 | .04367 |
| | | | | | | 1072 | 9.506 | | Lost |
| | | | 46.589 | | | Feces | | | |
| | | | | | | 157 | 33.668 | | .04367 |
| 875 | 20.46 | 400 | 25.48 | | | Urine | 32.452 | 63.269 | .06583 |
| | | | | | | 1220 | 5.529 | | .00514 |
| | | | 45.94 | | | Feces | | | |
| | | | | | | 97.2 | 37.981 | | .07097 |
| 1421 | 48.339 | | | 2.706 | .600 | Urine | 16.046 | 30.601 | .03644 |
| | | | | | | 810 | 7.942 | | .00353 |
| | | | | | 48.939 | Feces | | | |
| | | | | | | 136.7 | 23.988 | | .03997 |
| 1368 | 44.049 | | | | | Feces | | | |
| | | | | | | 127.9 | 10.206 | | .00289 |

The dog's weight at beginning of each period was respectively 16.1, 16.1, 15.876; 4th period beginning 16.102, close 16.329; 5th beginning 16.20, close 15.97 kilos.

expect that nascent oxalic acid, so to speak, freed in the intestine, would from the presence of calcium cause a large increase in the feces of oxalic acid, a supposition flatly contradicted by the results, an increase only of from 1.6 to 5.1 and 3.5 mgms. Nor can it be said that elimination of oxalic acid to any great extent takes place by means of the intestinal mucous membrane, as is true of many other metabolic products, nitrogenous matter for instance being eliminated during the entire period in death from fasting.

The rapid rise in both urea and oxalic acid while feeding with gelatine and glycocoll shows that while the nitrogenous group in the molecule is converted to the form of urea, the carbohydrate group appears as oxalic acid. In addition, this must have been absorbed and conversion have taken place by means of the cell since it is a process, partially at least, of oxidation which is the function of the cell. This fact is probably not only true of gelatine but also of other proteins which, as before stated, Karl Spiro has shown to contain glycocoll, but in a lesser degree because they contain less of this substance. Of course we know that glycocoll pairs with benzoic acid to form hippuric acid, and when no glycocoll is provided Hugo Wiener (Archiv. f. eperiment. pathol. and pharmakol. 40, 313–325) has shown that rabbits have a reserve supply of this substance equal to .3276–.3496 grams per kilo weight. Conversely when no benzoic acid is present it is evident that the glycocoll is chiefly eliminated as oxalic acid. This reserve can come from no other source than the breaking down of the albumen molecule containing glycocoll, and if oxalic acid comes from the same, we have in the former a measure of this decomposition, so that oxaluria, so called, which of course must be demonstrated only by quantitative analysis and not by mere precipitation, accompanied by various nervous manifestations, may have some basis in fact, not as a cause of these mani-

festations but as a result and measure. Finally we may justly draw the following conclusions :—

(1) That oxalic acid is derived from the carbohydrate group in albumens, presumably through the intervention of glycocoll by a process of splitting through which the remainder is converted to urea, a vital process though imitated by strong oxidizing agents.

(2) Oxalic acid may also be derived from uric acid by fermentative action and oxidizing agents, and perhaps also by vital processes.

(3) That oxalic acid, present in many organs of the body, practically absent in the feces of animals fed on non-oxalic acid containing foods, yet ever present in the urine after long periods of fasting, may rightly be called a metabolic product and rightfully take its place with acetone, lactic acetoacetic and oxybutyric acids as a measure of abnormal retrograde metamorphosis.

ARTICLE XL.

# DEMONSTRATION OF A MODEL OF THE ABDOMINAL VISCERA.

By THOMAS DWIGHT, M.D.,
OF NAHANT.

JUNE 12, 1901.

# DEMONSTRATION OF A MODEL OF THE ABDOMINAL VISCERA.

A TECHNICAL discussion of the various investigations carried on in the Anatomical Department of Harvard would probably have no great interest to the body I have the honor of addressing. I prefer to speak rather of principles and to show a model, which, apart from any intrinsic interest it may have, is an illustration of those principles. As the Society knows, I have devoted myself for many years to the study of variations and anomalies. I have striven in my teaching to popularize the fact that all men are not, so to speak, cast in the same mould, and that the study of the variations is by no means a matter of merely scientific interest, but one of real value to the practitioner. A notable point is the change in methods and scope of anatomical teaching. For instance, the study of the details of arterial anastomoses and of nerve distribution, though not neglected, are much less considered than they were a dozen years ago; while on the other hand the study of the viscera is commanding a continually increasing attention. The method of frozen sections, which I believe I introduced into this country, the making of reconstructions from the same, and the use of formaline as a hardening agent, have enabled us to get a knowledge of the real shapes of viscera, of which anatomists thirty years ago knew little more than was known after the rise of modern anatomy. One or two general conceptions of great value have become by this method

very familiar. One is the total absence of free spaces between organs, the other the fact that in growth one organ is moulded upon another, and more remarkable, that it is the hollow organs that seem to determine the shape of the solid ones and not the converse. Certain glandular organs which in the adult seem very resistant, have in fact grown by forcing their way in between others in the line of least resistance.

Whoever searches diligently for anatomical variations is sure to be rewarded not only by finding what he looks for, but occasionally by coming across remarkable anomalies. Among the curios I have found I will merely point to this case of total absence of the right kidney, to this of the aortic arch crossing the right bronchus, and to this of absence of the inferior vena cava below the diaphragm.

I now come to the task of showing this model of the abdominal viscera, enlarged three times on a linear scale, the work of Mr. James H. Emerton. For its proper understanding the condition of the body and the method of hardening must be described. The body was that of a young and slender man. The thorax had been opened and used for the demonstration of the viscera within it, but the walls of the abdomen, including the diaphragm, were quite intact. As the body had already been injected with a preservative, and the arteries with a colored mass, it was not possible to inject the formaline. I resorted, therefore, to pouring a solution of it down the gullet and to having it carried onward by gentle manipulation of the abdomen. This was done so thoroughly that the viscera were most perfectly hardened. The stomach and the intestines were, of course, distended, but happily not distorted, so that the model shows the condition they present when full to the utmost. The model was made from a series of plaster casts taken by Mr. Emerton. First, one was taken of the surface of the upper portion of the abdomen, after removal of the walls

and of the diaphragm. The liver was then removed and the process repeated. Next, the stomach was taken out and so on. From these plaster casts this large paper model was prepared. The viscera shown are the liver, the stomach, the duodenum and pancreas, the spleen, the kidneys with the suprarenals, and the traverse part of the colon with the two flexures. The aorta, the cœliac axis with its branches, the trunk of the superior mesenteric artery, the inferior cava, the portal vein with a part of the two large veins which chiefly form it are also shown.

The model as a whole (Fig. 1) gives a good idea of the compactness of the arrangement of the parts. A striking feature is that the liver extends across the entire breadth of the abdomen. I am inclined to think from the study of

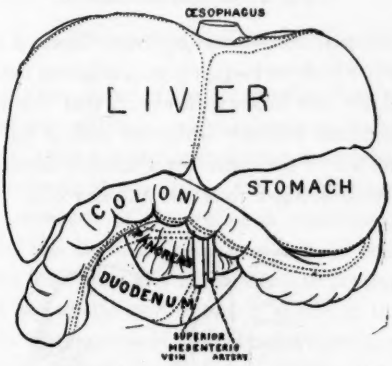


Fig. 1. Front view.

other specimens that this is by no means very uncommon. There are different forms of livers. This one (Fig. 2) has a long and thin left lobe, in marked distinction from another form of which I have a cast, in which there is great depth and solidity of the right lobe. Very probably the amount of liver tissue in the two is about the same. The moral is that it is not easy to determine clinically whether or no a

liver is enlarged. On removing the liver we obtain a good view of the stomach (Fig. 3). It shows at a glance that the lesser curvature is nearly vertical, even in a distended

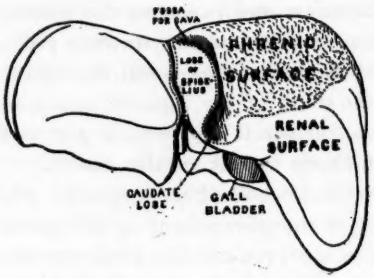


Fig. 2. The liver from behind.

stomach, till near the *antrum pylori*. There is a line across the stomach which corresponds to a ridge on the liver fitting into it. I do not believe, however, that the ridge is the cause of the line, because when we look at the back of the stomach we see at the same level another depressed line, if anything more marked than the anterior one. This shows

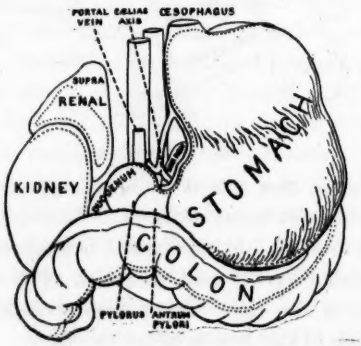


Fig. 3. Front view, the liver being removed.

the tendency of the organ to be divided into an upper and a lower half, an exaggeration of which produces "hourglass-stomach." This is seen also on the left side of the model (Fig. 4). The model of the stomach is moreover interest-

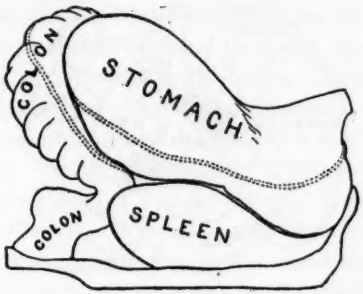


Fig. 4. Seen from the left, the liver being removed.

ing as showing the shape of the organ when fully distended. It disposes of the old idea, which, however, has been contradicted, that when the stomach dilates, it swings forward, the greater curvature advancing and moving towards the right. The entrance of the gullet is much nearer to the front than to the back. The upper half of the stomach dilates chiefly backward and the lower half forward, as the side view of the model well shows. While it is probable that owing to the absence of the thoracic viscera, the stomach and colon pressed the diaphragm upward to a degree that would not otherwise have occurred, it is clear, to me at least, that the stomach cannot, under normal conditions, really occupy the place it would seem to when shown by internal illumination.

Before proceeding to the deeper parts of the preparation it will be best to notice the colon (Fig. 1), which is in a highly distended condition. It moulds itself in a festoon under the right lobe of the liver and along the greater

curvature of the stomach. The position of the splenic flexure in a pocket between the spleen, stomach and left kidney is worth noting. The part under the liver com-

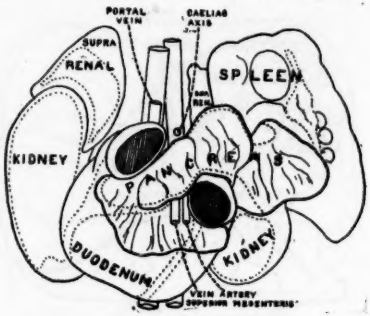


Fig. 5. Front view, the liver, stomach and colon being removed.

pletely compresses the fundus of the gall-bladder. The double line along the colon represents the attachment of the great omentum which is seen on the under side of the colon on the right, and gradually crossing it to reach the upper surface on the left. The extreme distention of the stomach of course modifies the position of the colon, which if the stomach were empty would be in much more extensive relation with the liver and would encroach on the diaphragm.

On removing the stomach, liver and colon (Fig. 5), we come to the, to me at least, most interesting part of the model. We see the spleen, nearly the whole of the right kidney, a part of the left, both suprarenal capsules, pancreas and duodenum.

The spleen, like a large proportion of spleens, is a good deal removed from the conventional type of the organ. Cunningham's basal surface is hard to make out. About the hilum there is a tendency to excessive lobulation, which makes the organ all the more peculiar, though this tendency in itself cannot be called abnormal.

The shape and position of the suprarenals are very well shown, but require no special comment.

The duodenum is very instructive. The four parts are readily recognized. I could have wished it had been different, for I published some time ago some observations on a considerable series, by which I showed that the third part of the duodenum crosses the aorta (which is considered the normal relation) only once in four cases. Here it crosses it most unmistakably. In the same paper I denied that the fourth, or ascending part on the left is ever in front of the left kidney. I cannot deny that it is here in front of that organ, though to but a slight extent. I conceive that the latter peculiarity is in this case due to the extreme distention of the gut. In any case the model shows the relations as they were in this case; but I consider the condition exceptional. The pancreas is of all the organs the most remarkable. Those not used to hardened specimens would probably not hesitate to declare it absolutely irregular. For my part I hesitate to say just how irregular it may be. In fact I am convinced that some of the features that seem irregular to those trained only in old-fashioned anatomy are, though in a less degree, not uncommon but even normal. It shows admirably how this organ develops by moulding itself on its neighbors. We see how it tends to embrace the end of the first portion and much of the second of the duodenum, curling round it behind as well as in front. The most remarkable thing about it is the great development of the head, which overlaps not only the third part of the duodenum, but extends across the fourth to its very left border. It is, I am sure, very common, if not the rule, for the head more or less to overlap the duodenum; but I have seen nothing like this excessive extension of it to the left. Clearly, however, like the liver the pancreas grows as it can, and the essential in its development is to find room for a certain amount of pancreatic tissue rather than to have this in a definite, conventional shape.

This is one of the vital facts that modern anatomy is emphasizing. For these studies we need many observations. If we show nothing but the range of variation we have done the clinician a real service.

ARTICLES XLI.–XLV.

# IN WHAT CASES OF DISEASE OF THE ALIMENTARY TRACT, NOT EMERGENCIES, SHOULD OPERATION BE ADVISED FOR RELIEF OR CURE?

## STRICTURE OF THE ŒSOPHAGUS.

By THEODORE DUNHAM, M.D.
OF NEW YORK CITY.

## GASTRIC ULCER.

By ARTHUR T. CABOT, M.D.
OF BOSTON.

## INTESTINAL CANCER.

By FRED B. LUND, M.D.
OF BOSTON.

## OBSTRUCTIVE DISEASE OF THE LOWER BOWEL.

By HENRY O. MARCY, M.D.
OF BOSTON.

## GALL STONES.

By MAURICE H. RICHARDSON, M.D.
OF BOSTON.

READ JUNE 11, 1901.

ARTICLE XLI.

# TREATMENT OF STRICTURE OF THE ŒSOPHAGUS.

By THEODORE DUNHAM, M. D.
OF NEW YORK CITY.

READ BY INVITATION.

I NEED not here discuss the pathology of strictures of the œsophagus. The accepted methods of treatment for both neoplastic and cicatricial strictures are familiar to you all. I think you will agree that with the methods thus far employed, some new method of dilating cicatricial strictures would be welcome if it should be rapid and comparatively safe.

During the past winter I have devised some procedures for the treatment of cicatricial stricture which have met with such perfect success, that I venture to ask your attention while I describe them.

The first requisite for dilatation of an œsophageal stricture is to get some instrument to pass through the œsophagus. In some cases all attempts to pass an instrument by way of the mouth fail. In a certain proportion of these cases after performing a gastrostomy it is possible to make an instrument engage in the cardiac orifice of the œsophagus and push it up into the mouth. But sometimes even this is not successful. I had such a case during the winter. I could pass an instrument neither from above nor from

below. I overcame the difficulty in the following way: The apparatus consisted of a glass of water, an ordinary drinking tube and a piece of black silk thread. I threaded the tube so that the end of the thread was at the mouth-end of the tube. The patient then drank water through the tube. The thread was washed along in the current of water, and as it disappeared up the tube more thread was fed into the glass of water. When several feet of thread had been thus washed down, I fished the lower end of the thread out from the stomach by passing a bent probe into the gastrostomy opening. This was entirely succcessful at the first attempt. On trying it subsequently, however, I failed because my patient, who was a child, had taken a dislike to the procedure and snarled the thread with his tongue, thus preventing its washing down.

This new difficulty I overcame as follows:—To a glass funnel I attached a piece of rubber tubing, and to this, by a piece of glass tubing, a small rubber catheter the tip of which had been cut off. A thread was washed down from the funnel until its end appeared at the end of the catheter. The rubber tubing was now pinched to stop the flow of water and thread, and the catheter inserted into a nostril until its end hung in the pharynx. A swallow of water was now allowed to flow from the funnel, and then the tube was again pinched. Water was thus intermittently fed down the œsophagus, the thread going with the stream. After several feet of thread had disappeared down the funnel I fished the lower end of the thread out through the gastrostomy opening as before.

Before trying to wash a thread down the œsophagus of my patient, I first tested the feasibility of doing so upon an artificial œsophagus. I have made a similar one to show you now. It is made of glass and rubber tubing. The funnel above may represent the pharynx. The channel passes from the neck of the funnel through a piece of rub-

ber tubing and a piece of glass tubing, and then by means of a perforated cork into a glass tube of large calibre. This is intended to represent a dilated pouch above a stricture. The exit below from the large tube is by a small glass tube which passes half an inch into the large tube, to represent the mouth of the stricture as lying at a higher level than the bottom of the pouch. Thence the channel is continued by a piece of rubber tubing, by a piece of glass tubing which takes a circular turn in a vertical plane and then makes a sharp and constricted lateral bend, and the channel finally ends by a rubber tube having a diameter of about one sixteenth of an inch. I will introduce a thread into the neck of the funnel and then keep the funnel filled with water and gradually feed in the thread. The thread is carried by the current of water through the whole system without difficulty. Should it be arrested at any point, a momentary pull will disengage it and allow it to pass on.

Having passed a thread from the nostril to the gastrostomy opening, my next hope was to dilate the stricture by using the method devised by Dr. Robert Abbe and used with perfect success in a case reported by him a few years ago. His method consists in putting the stricture upon the stretch by engaging in it a conical gum elastic bougie and then sawing the stricture by pulling back and forth a stout thread passing through the stricture. In attempting to use this method I first pulled through a double thread of strong silk. By means of one of the two resulting threads I pulled through a piece of stout linen fish line; by the other one I attempted to draw into the stricture a conical gum-elastic bougie having an eye at its tip to which the thread was attached. I was unable to make the bougie engage in the lower end of the œsophagus even when it was thus guided by a thread.

I then devised the wire-and-spindle bougies which I will show. Each consists of five feet of steel wire having a tiny

knob at each end and carrying at its middle a conical piece of metal. I had a series of these made, carrying spindles of different diameters, the smallest being No. 10 French and the largest No. 30.

These wire-and-spindle bougies I used in the following way:—The fish line was drawn through the œsophagus. The wire of a bougie was then attached to a thread and pulled through the œsophagus until the spindle was arrested at the stricture. One end of the wire and of the fish line now projected from the mouth, the other ends from the gastrostomy opening. To guard the soft parts of the pharynx and of the stomach I made two guide tubes which consist of aluminum tubing, one bent to pass into the pharynx behind the larynx, and the other bent to pass through the gastrostomy opening and up to the cardiac orifice. The wire and fish line were drawn through these tubes above and below. Tension was then made upon the wire above and the fish line was drawn back and forth. The wire began to yield and soon the spindle was heard and felt to click against the upper guide tube, showing that a way had been cut for it through the œsophagus. Larger wire-and-spindle bougies were then successively cut through in the same way, a No. 30 French being the largest I considered desirable as my patient was only three years old.

The foregoing procedures apply to cases in which it is necessary to open the stomach, because no instrument can be made to pass from above.

In those cases in which it is possible to pass an instrument from above, I need not remind you of the dangers of attempting to dilate by forcing down the usual conical bougies. To avoid these dangers I devised an instrument by means of which the idea of cutting the stricture with a string can be carried out by working entirely from above without opening the stomach. I have used this instrument with complete success this Spring and therefore venture to present it to you.

The instrument consists of a stout staff of whalebone to the end of which is screwed fast an olive-shaped piece of metal. This olive is pierced by two tunnels. One is a curved tunnel having its convexity towards the end of the instrument and its two ends opening opposite one another well forward of the greatest diameter of the olive. This curved tunnel is accomplished by making two borings which meet at an angle. The sharp crest where the borings meet is smoothed down by boring through a string covered with wet emery powder. Unless the metal is thus smoothed the cutting string will itself be quickly chafed in two.

The second tunnel begins at the base of the olive, to one side of the screw which attaches it to the whalebone staff, and, passing diagonally through the olive, ends at its tip. This tunnel I will call the guide-tunnel. The instrument is held to its course in the œsophagus by what I may call a guide-bougie. This consists of an urethral bougie cut off to a length of seven or eight inches. A steel wire passes through the lumen of the bougie. One end of the wire terminates in a metal tip at one end of the bougie. Where the wire emerges from the other end of the bougie it is soldered to a metal tip on the bougie and then extends beyond it some three feet. To shield the soft parts of the pharynx from the scraping of the string while cutting, I made a guard to hold the strings close to the whalebone staff. This guard consists of a handle to which are soldered two wires. These wires run along the whalebone staff and carry three wire loops encircling the whalebone, by means of which the guard may slide along the staff. The wires also carry two sets of lateral eyes in which the cutting strings may play two and fro. The strings are thus held away from the soft parts.

The method of using this instrument is as follows: an olive a little larger than the stricture is screwed to the whalebone staff. The two ends of the string which passes

through its string-tunnel are threaded through the eyes of the guard. If the patient be a child, he is best seated in the lap of an assistant, with the shoulders resting against a pillow in front of the assistant, and the head thrown back over the top of the pillow, the mouth held open by a gag. A guide-bougie is chosen of such size that it will readily pass the stricture. It is introduced until all but the wire has disappeared behind the epiglottis. The end must then be in the stomach. As no force is required in passing this guide-bougie, it cannot make a false passage. The wire of the guide-bougie is now threaded through the guide-tunnel of the olive and the olive slid along the wire until its tip comes in contact with the bougie portion of the guide-bougie. By pulling upon the wire and pushing the staff, the guide and olive become now virtually one instrument. As the guide is necessarily in the œsophagus the olive must follow it and cannot make a false passage even if a fair amount of force is used. One thing must be guarded against: kinking the wire where olive and bougie meet, by failing to direct the staff towards the œsophagus, while the olive is still in the pharynx. Attention to the position of the patient and proper handling of the whalebone staff will prevent this from happening. When the olive is arrested by the stricture, the guard is slid along the staff until its end is beyond the epiglottis, and then an assistant pulls the string two and fro, pulling parallel to the staff to make as little friction as may be. Moderate pressure is made upon the staff, which causes the olive to put the stricture on the stretch, and the strings cut a passage for it. As the olive descends the guide-bougie coils up in the stomach. Owing to the slight force required to coil the bougie in the stomach, the olive may not, when it has cut through the stricture, plunge suddenly into the stomach. It is not, therefore, safe to wait for a sense of lessened resistance as an indication that the olive has passed the œsophagus. The

safe plan is to mark the bougie at the point which should be at the incisor teeth when the olive is in the stomach. For a child of two years, ten inches should be right.

After cutting strictures by the foregoing instruments it is of course necessary that bougies be passed at intervals to guard against new contractions. I hesitated to pass the ordinary bougies for fear of making a false passage at the cut portion of the œsophagus. I therefore had made a set of hollow bougies which could be passed on a guide. They are ordinary gum-elastic bougies, with the ends cut off and a perforated conical metal tip screwed on. In my case, which had a gastrostomy, I pulled a wire from the stomach wound up through the mouth, and, using this as a guide, slid these hollow bougies down the œsophagus until their tips appeared on the abdomen. Subsequently I first introduced one of the guide-bougies such as is used with my stricture-cutter and, making sure that this had gently found its way to the stomach, I felt no hesitation in pushing down a No. 30 hollow bougie. This has been done at intervals of about a week. The gastrostomy wound has nearly closed spontaneously.

In the case operated entirely from above, the guide-bougie is also used to carry hollow bougies and maintain the dilatation. This patient is eighteen months old and a No. 27 French bougie is passed.

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## DISCUSSION.

Dr. S. J. Mixter, of Boston:—I am sure that every member present will feel grateful to Dr. Dunham for his excellent paper. His methods are ingenious and very successful. He has explained many difficulties in a very satisfactory and plain way.

Any one who has had any practical experience in the treatment of œsophageal strictures knows how difficult it is

to get through certain strictures where liquids will pass. Dr. Dunham's advice simplifies this matter very much, and renders the procedure less dangerous than formerly. This is especially true in the malignant œsophageal stricture, where we wish to put in a permanent tube. His advice about operating on the stricture from above, without entering the stomach, seems to me a brilliant idea.

I would like to suggest a slight modification that can be made with regard to the guide-bougies. As we know a pouch often forms above a stricture, and as this grows larger it generally dilates unequally, so that finally it is well to one side of the line of the œsophagus and the lower opening, through the stricture, is in the side of the pouch and not at its lowest point. If now a straight instrument is introduced it passes to the bottom of the pouch without engaging in the stricture. I have found that by making a bend in the bougie an inch or two from the tip, best done by means of a wire stylet in the centre of the bougie, the opening through the stricture may often be found and passed when a straight instrument will fail.

I often use a Symonds tube as a dilator, leaving it in place several days. As this allows liquids to pass, the pressure on the pouch is done away with and, in time, the muscular wall of the œsophagus regains its tone and the pouch disappears, making subsequent passage of instruments easy and safe.

ARTICLE XLII.

# A DISCUSSION OF THE INDICATIONS FOR OPERATION IN GASTRIC ULCER.

By ARTHUR T. CABOT, M.D.
OF BOSTON.

Our experience in the operative treatment of gastric ulcer has been considerably widened in the past few years, and yet I think little of practical value has been added to our knowledge of the indications for operation which were so excellently set forth by Leube and Mikulicz at the German Surgical Congress in Berlin in 1897. They well recognized the limitations of medical treatment in dealing with this troublesome disease, and we can add but little to the discussion of that part of the subject.

The changes which have taken place since then in our knowledge of this subject have been mainly in the line of improved surgical technique and consequently lessened danger from operative measures. As the risks of operation diminish, we have to frequently readjust our ideas as to the persistence with which medical treatment must be tried before resort is had to surgery.

Too often the question of operative interference in gastric ulcer has been postponed, not only until medical treatment has been long and evidently a failure, but even until the patient's condition has become desperate.

Let us see whether surgery offers a sufficient prospect of relief in this condition to encourage our medical brethren to call us earlier, while the patient still possesses the strength to endure a somewhat serious operation.

In order to properly present the indications for operation in gastric ulcer, it is necessary first to give a moment to the consideration of the different operations on the stomach which may be made useful in the various phases of this disease.

These are: —

1. Gastrotomy, including the excision of ulcers.
2. Gastroplication, or turning in of the stomach wall to close an ulcer that has perforated, or to strengthen the wall at a point where perforation is threatened.
3. Pylorectomy for the removal of an ulcerating pylorus.
4. Pyloroplasty for the widening of a pylorus contracted by ulceration.
5. Gastro enterostomy to provide a short cut into the intestine from a stomach whose motility is interfered with by ulceration.

Gastrotomy and the excision of ulcers has so evident an application in cases where the ulcer can be located and reached that nothing need be said in explanation of it for the purpose of this discussion. The same is true of gastroplication which is plainly the proper procedure for closing small perforations. Pylorectomy, too, needs no especial consideration here, as it simply amounts to the mechanical removal of the diseased portion when situated at the pylorus.

When we come, however, to pyloroplasty and gastro enterostomy, we have operations which have a wider range of usefulness than any of those mentioned. Their utility in relieving obstruction at the pylorus due to cicatricial contraction is plain and readily understood, but it has been further abundantly shown that these operations, particularly gastro enterostomy, have a very decided influence in pro-

moting the healing of ulcers even when there is no evidence of pyloric obstruction. It is to be remembered in this connection, however, that even when no obstructing lesion is discoverable by inspection and careful examination at the time of operation, we are still not justified in concluding that the pylorus offered no harmful resistance to the passage of the stomach contents into the intestine. For we know that spasmodic contraction of the pylorus plays a prominent part in some gastric conditions and there is reason to suspect that it must be reckoned with far oftener than has hitherto been supposed. If such spasm has been of long duration, a thickening of the muscular ring about the pylorus may be recognizable, but a moderate spasm excited temporarily and intermittently by the irritation of food may play an important part in prolonging and aggravating an ulcerative process in the stomach wall and yet lead to no appreciable muscular hypertrophy. Whether we attach much importance to pyloric spasm or not, it is certain that a gastro enterostomy is usually followed by a great amelioration of symptoms and by the disappearance of the excess of hydrochloric acid which plays so important a part in the production of gastric ulcer.

It is also of great significance that a gastric hemorrhage is frequently brought to a stand still by a gastro enterostomy. This result follows the operation so immediately as to suggest that the over active peristalsis of the organ is quieted as soon as an ample drainage is afforded for its contents.

Mr. Mayo Robson lately reported a recent case of his in which after repeated severe hemorrhages he operated during an attack of bleeding and had an almost immediate cessation of it.

The mere correction of the hyperacidity could hardly have produced so immediate an effect, but the cessation of the active struggle of the stomach to free itself might well fol-

low directly on the operation which gave the contents free exit, and this would seem to be the most plausible explanation of the quick effect produced in these cases.

This brief review of the operations applicable to the relief of gastric ulcer shows what a great practical advance was made in the discovery of the efficacy of gastro enterostomy in the treatment of this affection. When the surgeon approached these cases with the feeling that he must find the ulcer and operate directly upon it by excision or otherwise, he might well feel deterred by the doubts whether in the first place he could find the ulcer, and secondly, whether if found it would be accessible to safe operation. It is no wonder that these doubts, together with the very great risks of the operation, prevented early resort to surgery and kept the patients under medical treatment until their strength was so sapped that surgical interference was indeed a forlorn hope.

With gastro enterostomy added to our repertoire, however, we can feel that if we do not find the ulcer accessible to safe removal, or even if we cannot find it at all, we may still afford relief to our patient by an easy and comparatively safe operation. The feeling of confidence which this knowledge affords has already begun to bring these patients to operation earlier, and the immediate improvement in results that has followed, gives great encouragement for the future

After this brief outline of the present position of the surgery of ulcus ventriculi, we are come to the proper subject of this paper, namely, the consideration of the indications which should lead us to conclude that a given patient is less likely to recover under medical treatment than with the aid of surgery.

To begin with the conditions most evidently and indisputably demanding interference, it is unnecessary, I think, to urge the need of an immediate operation when symptoms of perforation appear.

It is true that occasionally a case of perforating gastric ulcer recovers under medical treatment. This occurs in a certain proportion of those rare cases in which gas alone escapes from the stomach into the peritoneal cavity. So exceptional a possibility cannot be used as an argument for delay, however, as it is far more than counterbalanced by the fact that the mortality attending an operation done inside of twelve hours of the perforation is only about one-half that expected in cases where operation is delayed to twenty-four or forty-eight hours.

In no other abdominal condition, except perhaps internal hemorrhage, is the success of interference so dependent on its promptitude. This is of so great importance that in cases of gastric ulcer in which acuteness of pain and tenderness indicate that the ulceration is approaching the peritoneum, it is well to make preparations which will insure a quick operation if the need arises. Early recognition of such a warning will save time when the patient's chances of recovery are slipping away at the rate of many percent an hour.

Dangerous or persistent hemorrhage is another well recognized indication for operation. The fact, however, that gastric hemorrhage is very rarely severe enough to cause speedy death and that many cases in which this is a prominent symptom recover when treated medically, must be taken into account and must often lead us to choose a conservative course. A consideration of the cases in which hemorrhage occurs in gastric ulcer would seem to show that in a rough way we may divide them into two classes.

In the first class we would place those cases occurring before thirty years of age, and in the second class those appearing in later life. In the younger patients the hemorrhage is usually from the opening of some vessel of the stomach wall by the ordinary round ulcer. This form of ulcer so common in young women often leads to hemorrhage in them, but very rarely leads to fatal hemorrhage.

This was pointed out by Hood, who found at Guy's Hospital for the twenty years between 1870 and 1890 no case of fatal hemorrhage occurring in a young woman, and Greenough and Joslin in their study of the cases at the Massachusetts General Hospital found that the same was true at that Hospital between the years of 1888 and 1898. An attempt has been made to explain this immunity from danger by the fact that women bear hemorrhage better than men, but it seems to me that the more plausible explanation lies in the fact that these early ulcers are more superficial and the hemorrhage comes from the comparatively small vessels of the gastric mucous membrane.

A hemorrhage occurring in the second period, after thirty years of age, is on the other hand more likely to be the result of chronic ulcer, one of those slowly extending ulcerations which penetrates deeply into the walls of the stomach and threatens the large vessels lying near the serous surface and which in extreme cases may lead to perforation of arteries in neighboring organs (pancreas, liver or spleen) to which the stomach has become adherent. We should feel, therefore, that the danger of fatal hemorrhage is greater in later life and especially where the symptoms point to a long existing lesion of the stomach.

Under such circumstances, a tendency to slight recurring hemorrhages should act as a warning to us that the ulcer is progressing in a possibly dangerous direction.

As has been said, excision of the ulcer or ligature of the bleeding vessel is often impossible on account of its position or because it cannot be found. In chronic ulcers, too, when they have penetrated into surrounding organs, excision cannot be practised and constriction of the vessels by a surrounding stitch is an uncertain and somewhat dangerous procedure. The question then naturally arises,—To what extent may we rely upon gastro enterostomy to bring the hemorrhage to a stand still? The evidence seems to show

that in a large proportion of cases it is efficacious. That it should sometimes fail is to be expected. Körte reports two cases of fatal hemorrhage from ulcers eight and twelve days after gastro enterostomy. The possibility of this occurrence would certainly lead one to excise an accessible ulcer rather than trust to gastro enterostomy to stop the hemorrhage. In case, however, the ulcer cannot be safely removed, we are justified in expecting a usually good result from gastro enterostomy.

With an understanding of the different characteristics of the two forms of ulcer it will be readily seen that in the case of hemorrhage from an ulcer occurring early in life, operation may be more properly deferred than in an ulcer appearing or persisting after thirty years of age. The diagnosis of a chronic and progressive ulcer, as distinguished from an ordinary round ulcer, is still further suggested when the patient has had a history of past attacks of dyspeptic character. When a frequently recurring hemorrhage appears as a leading symptom in the course of a chronic gastric ulcer, it is so sure a sign of impending danger that it should be regarded as a clear indication for operation. In these cases, even when a large vessel is not ultimately opened, intermittent hemorrhage gradually brings about a condition of anæmia which favors the extension of the ulcer rather than its healing, so that under these circumstances a cure is practically hopeless except by operative measures. These are the patients who often supply the surgeon with his most brilliant successes.

Hitherto we have been considering the treatment of complications of gastric ulcer.

We now come to the question of operative measures for bringing about the healing of the ulcer itself, and to a consideration of the evidence which should convince one that medical treatment has proved unavailing in a particular case and that resort must be had to surgery.

As a preliminary to this study it may be well to look at the mortality of gastric ulcer *per se* and compare this with the mortality attending the various operations for its relief.

Attempting this comparison, we are at once met with the difficulty of establishing any fair mortality rate for the disease. Various observers have set this at widely different figures, ranging from 15 to 50%. This divergence is largely due to the difficulty of following patients to the end. The lower figure, 15%, does not sufficiently take account of the morbid conditions left after the healing of an ulcer and of the subsequent threat to life that these entail.

Mikulicz in endeavoring to properly allow for these subsequent dangers which belong to the history of ulcer, inclines to the belief that from 25 to 30% fairly represents the proper mortality of the disease.

Notwithstanding all that has been written on the subject of gastric ulcer, it is surprising to find that otherwise good observers have not studied the end results of their cases, so that with a disease showing so great a tendency to recurrence, their reports are of little value. We are fortunate in having one notable exception to this rule in the very full study made by Greenough and Joslin of the ulcer cases occurring at the Massachusetts General Hospital between the years of 1888 and 1898. They find that out of ninety-two patients who left the Hospital cured or relieved, thirty-eight had a recurrence of their trouble and twelve died, eight of these deaths being undoubtedly due to the gastric condition. This leaves but forty-two out of ninety-two who remained well an average of five years after their recovery from gastric ulcer. According to their investigations then the proportion of patients who were cured is between 40 and 50%. The greater proportion of patients either died or suffered a recurrence of the affection.

Investigating now the risks of the various operations, Mikulicz found them as follows:—

1. Resection of the ulcer, 27.8% mortality.
2. Gastro enterostomy, 16.2% mortality.
3. Pyloroplasty, 13.2% mortality.

Adding all of the cases together, he found a mortality of 16.1% for all operative interference in this disease.

Setting this figure against 25 to 30%, the mortality of the disease, he draws conclusions decidedly in favor of operation. Could he have had the figures of Greenough and Joslin his arguments would have been even more conclusive.

The matter is not so easily settled as this, however, unless it can be shown that after operation the cure is lasting. Unfortunately this is not the case. We know that recurrences occur after operation, but we have no data to show how frequent these recurrences are.

It may well be doubted whether they are not of sufficient frequency to annul the fancied advantage of the above figures quoted from Mikulicz's per centages.

Fortunately the three years which have passed since Mikulicz made his calculations have seen such great advances in the surgical technique and consequently in the safety of gastric operations that we may properly consider the surgical risk as very decidedly lower than he did.

The most modern statistics give the death rate of gastro enterostomy as distinctly below 10%. Mr. Mayo Robson has even reduced his mortality, after this operation, to approximately 5%.

This improvement in statistics is largely due, no doubt, to the fact that the patients come to operation earlier.

As soon as an ulcer has made its chronic or its progressive character known and has refused to yield to proper trial of medical treatment, then is the proper moment for surgery to step in, and by affording rest and free drainage, as it were, of the stomach to favor a solid healing of the ulcer. By such a course we might hope to cure many ulcers which,

neglected, run on to an intractable condition which finally resists a late, almost despairing attempt at surgical relief and adds one more failure to the score against gastric surgery. This is no fanciful picture, but a fair statement of the course run by many cases in the near past. To make such cases rare in the future, it is important that in the first place the clinical physician shall make a closely studied and accurate diagnosis of the existence of ulcer and that he shall satisfy himself as to whether he is dealing with a first attack or with a recurrence of a formerly existing condition.

If he satisfies himself that he is dealing with a chronic, recurrent ulcer, especially if spasmodic pains and vomiting suggest an obstructed pylorus, he should at once suggest the propriety of operative interference and have the question thoroughly discussed.

The consulting surgeon called to such a case would be justified in advising at once proceeding to an exploratory operation with the intention of relieving the condition found in the manner best suited to the exigencies of the case.

If, on the other hand, the clinician is satisfied that he is dealing with a first attack of ulcer, he will treat the case medically with a proper watchfulness for complications. If the case drags out into a chronic course, with ups and downs, but never with complete recovery, it will again be well to consider the question of surgical interference.

When such a case recovers fully from the first attack, the patient or his friends should be warned of the dangers of recurrence and of the need of having good advice if such recurrence occurs.

If such patients, both those treated medically and those operated upon, can only be followed up and their subsequent history recorded, we shall presently obtain light that will enable us to see clearly the exact values of our results.

I will close with a condensed statement of the present indications for surgical treatment:—

1. Acute hemorrhages should rarely be treated by operation, the results of interference have not been good, while the results of medical treatment have been satisfactory. When, however, a hemorrhage frequently repeats itself even if severe in amount, it will demand operative treatment as soon as its recurrent character is plain.

2. Small frequent hemorrhages, threatening anæmia, give a clear indication for operation.

3. Perforation of the stomach, either acute with general peritonitis, or chronic with surrounding adhesions and perigastritis, demands instant operation.

4. When an ulcer runs a chronic course with a strong tendency to recurrence, and gradually diminishes the patient's capacity for work and for the enjoyment of life, an operation is indicated, especially when the patient is so situated as to be dependent on his daily work for support and unable to closely regulate his diet.

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## DISCUSSION.

Dr. F. B. Lund, of Boston:—I have had the opportunity of operating on two cases of gastric ulcer, one twelve hours and the other thirteen after perforation, and I was very much impressed by the prompt recovery in both cases, especially the last case. The ulcer was situated on the anterior surface of the stomach. The percentage of recoveries in such cases after operation during late years has been over 80.

My second case, that of a young woman, emphasizes the fact brought out by Dr. Cabot of the necessity for operating promptly. In this case, just before and after perforation took place, her symptoms were very severe. It would have been better had she been operated on before her symptoms became so very urgent.

In the case of chronic ulcers the affected part is surrounded by a hard fibrous tissue. These cases look very much like malignant disease. Last winter I operated on such a case situated on the lesser curvature of the stomach. The patient, a man, had for about a month before the operation repeated hemorrhages. When I cut down on the part I found the ulcer situated on the lesser curvature, in a bad place for operation. I took out a large piece of the stomach. When the walls were brought together it narrowed the stomach to about the size of the upper intestine. This man made a first class recovery, and was soon able to digest fatty and other ordinary articles of food. In this case I believe that no medical treatment would absorb the fibrous tissue and heal the part. My experience is not large in this class of cases, but such as it is would induce me to advise operation in many of them, and I can testify to the good results that can be obtained from a surgical standpoint in cases of this sort.

ARTICLE XLIII.

# CANCER OF THE INTESTINE.

By FRED B. LUND, M.D.
OF BOSTON.

The history of the majority of patients who are subjects of cancer of the intestine is a most unsatisfactory one and seldom redounds to the credit of medical science. Certainly those who see these cases as surgical consultants are generally abundantly impressed with their unfavorable side. We are asked to give surgical aid to patients suffering from the frightful emergencies in which this disease terminates, such as general peritonitis from perforation of the growth, paraintestinal abscess, or complete occlusion of the bowel from contraction of an encircling cancer. These grave emergencies admit in general of nothing more than palliative operations; radical measures, such as resection of the growth, are out of the question, and the immediate mortality of even palliative surgery is very high.

My own operative experience consists of only three cases of cancer of the intestine, although I have been able to follow a considerable number of cases in the practice of colleagues. One was a general peritonitis from perforation of a stercoral ulcer of the cæcum, in an old woman whose bowel was completely obstructed by cancer of the sigmoid, and who died four days after an artificial anus was made in the cæcum; one was a patient with localized abscess from perforation of a

cancer of the cæcum, who died three weeks after the abscess was drained, of peritonitis due to a second perforation; the third was a left iliac colostomy for complete obstruction of a cancer of the sigmoid, which was found to be situated so low in the pelvis and to have so extensively involved the peritoneum and glands that resection was out of the question. This patient lived in comfort for a year after the operation, a truss controlling the fæcal current perfectly. Such histories as these are not uncommon in cancer of the intestine, and suggest the advisability of an inquiry as to whether they might not have been improved. A positive answer may be found in the history of two of the cases, which had been suffering for two and a half years and three months respectively from symptoms which ought to have indicated exploratory laparotomy. The symptoms of emaciation, increasing constipation, perhaps alternating with diarrhœa, with localized gurgling of gas and intestinal discomfort, in a patient past middle age, ought to indicate exploratory laparotomy whether a tumor is felt or not,— for the tumor may be so situated, say deep in the right or left hypochondrium, as not to be palpable before it has gone too far to admit of radical treatment. If we wait for a definite diagnosis in chronic as well as in acute abdominal affections, we all too frequently sleep through the period where efficient therapeutics are applicable, to suddenly wake and find our patient the victim of a general peritonitis or acute obstruction, or the subject of a generalized abdominal cancer so extensive as to be far beyond surgical aid. In the rare cases where a tumor is so situated that it is felt early in the disease, there ought to be no question regarding immediate laparotomy; but unfortunately, even under these circumstances, the time is frequently wasted in waiting to make an absolute diagnosis.

There are several reasons why the diagnosis in intestinal cancer is frequently made too late in the disease, some of

which are the fault of the attending physician and some of which are not. The frequently insidious onset, the vagueness and elusiveness of some of the early symptoms, too often, instead of stimulating the medical attendant to a careful investigation with the possibility of intestinal cancer in mind, lead to a careless slurring over of the symptoms, and a falsely optimistic view of the patient's condition, which is much easier than an alert appreciation of the possibilities. The doctor does not like to tell his patient that he suspects a cancer until he is absolutely obliged to; and for this inadequate reason he puts off exploratory laparotomy, or even other simpler means of diagnosis, till after complications arise which make certain both the diagnosis and the death of the patient. Certain patients, particularly in the lower walks of life, will not consult a physician, or will resort to the aid of quacks or Christian Scientists until the onset of peritonitis or obstruction leads them too late to seek medical aid. The cases will be especially frequent in hospital practice, and for their demise the medical attendant cannot fairly be held responsible.

But in the cases which resort to him early in the disease, with vague or uncertain intestinal symptoms, including loss of weight, in patients past middle life, let him never forget the possibility of intestinal cancer, nor omit to carefully exhaust all means of diagnosis at his disposal, unless he is willing to be responsible for the frequently rapid progress of the trouble to an incurable stage. He may attempt to console himself with the thought that the results of surgical treatment in this disease have so far not been brilliant; but the answer to this is that the chief reason for the poor results has been the lateness with which the diagnosis has been made, and the case turned over to the surgeon.

Were I to answer the question given me to discuss, "In what cases of intestinal cancer, not emergencies, should operations be advised either for relief or cure," I should say, in

all cases in which an early diagnosis or even a probability has been established of the presence of intestinal cancer, exploratory operation should be performed with a view to removal of the growth. As far as we know the only possible cure of an intestinal cancer consists in its early removal, and even palliative measures, such as intestinal anastomosis or colostomy, are much more safely performed in the absence of the grave emergency of obstruction, and before the patient has been too greatly weakened by the advance of the disease. Absolute localization to a limited portion of the intestine, without extensive glandular involvement, in a patient in good condition, is requisite for successful treatment of an intestinal cancer. These conditions demand not only early diagnosis, but frequently exploratory operation in the absence of exact diagnosis.

Before discussion of the symptoms on which the diagnosis may be made, it will be wise to briefly enumerate certain anatomical and pathological characteristics of the disease upon which its clinical symptoms depend.

Cancer of the intestine occurs most commonly in the fourth to the sixth decade of life and is by far the commonest cause of chronic intestinal obstruction. It is about three times commoner in males than in females. It not infrequently occurs in the third decade, however, and as is the case with cancer is constantly being found in younger patients than we have a right to expect it.

The large intestine is almost invariably the seat of the disease.* Leaving out the most frequent location of all, the rectum, which is to be the subject of a special paper, cancer of the bowel has the commonest sites in the cæcum and sigmoid flexure, a fact to which the explanation has been suggested that these are subject to more constant irritation from the presence of fæcal contents, the cæcum being

* Körte found in 58 cases, 54 of large and four of the small intestine, three of the latter involving the duodenum.

the depot of detention for the fæces entering the large from the small intestine, and the sigmoid the reservoir in which they are held to await defecation. Next in frequency the cancer is found on the transverse colon, the splenic flexure, the hepatic flexure and the descending colon. Disturbance of intestinal function and obstruction appears earlier in cancer of the lower part of the bowel, because the intestinal contents are most fluid at the cæcum and become more solid and easily stopped as progress is made toward the sigmoid region. The common cancer of the sigmoid is the small, hard, scirrhous, contracting type, and owing to its physical characteristics the tumor of the sigmoid is apt to present the following peculiarities:—its small size makes it difficult to palpate, and it may be palpable at one time and not at another, depending upon the distention of the intestines surrounding it, especially the dilated portion above it. If situated in the lower part of the sigmoid or on the coil which hangs into the pelvis, it may be palpable by rectum, and not through the abdominal wall. The sigmoid having a long mesentery, it will be movable for a considerable period, and even after extensive secondary infiltration of the glands has taken place. Attacks of obstruction will appear early, owing to the scirrhous, contracting character of the growth. The long mesentery will favor resection.

Cancer of the cæcum is more apt to present the softer glandular type, the adeno-carcinoma; it is frequently situated for a long time upon the wall of the cæcum without encircling it, and therefore attains considerable size and is often easily palpable, long before obstructive phenomena are caused. It is less movable than sigmoid cancer, having a short, or no, free mesentery. Cancer of the hepatic and splenic flexures is usually of the obstructive type, and owing to the position of these flexures deep in the abdomen under the ribs, is difficult to palpate before it has attained considerable size. It is very slightly movable. Cancer of the

transverse colon, however, owing to the length of its mesentery, is frequently movable. It therefore becomes evident that the mobility of a tumor is no gauge of its operability. Even slight mobility of a cancer of a fixed portion of the intestine, such as the ascending colon or cæcum, would indicate a probability that the tumor was in an early stage and could be successfully resected, while slight fixation of a tumor of the transverse colon or sigmoid would indicate such adhesions to neighboring organs or involvement of the mesentery by cancer that the growth would be inoperable. We must not forget, however, that it is impossible to determine whether a tumor is operable or not before the abdomen is opened, or we shall condemn certain cases which might have been relieved. A favorable characteristic of intestinal cancer is the fact that the growth frequently remains a long time without secondry involvement of the mesenteric glands or of the liver, and that even in patients dying from intestinal obstruction the pathologists find tumors which might have been successfully removed. Certainly such pathological findings as these are a reproof to the clinicians, and ought to stimulate us to renewed efforts at early diagnosis of these conditions.

*Differential Diagnosis.*—We have to consider sarcoma, tuberculosis, benign tumors, cancer or tumors of other organs, fæcal impaction; and in case of obstruction without palpable tumor, benign scars and tubercular stricture.

Primary sarcoma of the large intestine is rare, occurring eighteen times in 426 cases of malignant disease of the large intestine collected by Bovis.* Sarcoma of the large intestine generally occupies the cæcum, ileocæcal valve or vermiform appendix.

Tuberculosis of the large intestine is also rare and is generally located at the cæcum. Points of value in the

* Bovis, Revue de Chirurgie. Par. 1900. XXII. 49-99.

diagnosis from cancer of the cæcum are the fact that the tubercular growth usually causes general thickening and infiltration of the cæcal wall, which feels boggy and doughy. In cancer we get a localized tumor, the rest of the cæcum being normal in feel. Tuberculosis of the cæcum rarely causes obstruction until very late in the disease, while cancer of the cæcum, though here it becomes obstructive later than in other parts of the intestine, notably the sigmoid, causes obstruction earlier than tubercle. The youth of the patient, the presence of active or latent tuberculosis in other parts of the body, and presence of tubercle bacilli in the stools may be valuable points in the differential diagnosis.

Obstruction of the intestine by tubercle is so rare that Erdheim,* in 566 cases of tuberculosis of the intestine, found only eight times a single, and once a double stenosis. Stricture of the intestine from tuberculosis comes on late in the disease, being due to the contraction of cicatricial tissue resulting from ulcers. The small intestine containing fluid contents, a very high degree of stenosis is necessary to produce obstruction, which is another reason for the lateness of appearance of that symptom. A tumor is rarely felt, owing to the small intestine being more difficult to palpate than the large.

Other rare causes of stenosis are benign tumors and contraction from the scars of benign (not tuberculous or malignant) ulcer.

When we have palpable tumor we have to think, in addition to tuberculosis, of chronic inflammatory processes and actinomycosis. Cancerous growths have been mistaken for movable kidney or renal tumors, and in case of the hepatic flexure and ascending colon, for tumors of the stomach. In sigmoid tumors in the female pelvis we must consider disease of the adnexa.

* Weiner Klinischer Wochenschrift, 1900. XIII. 79–83.

Tumors due to fæcal impaction may be distinguished from cancer by the cautious use of cathartics and enemata. The accumulation of hardened fæces above a cancerous tumor may deceive as to its apparent size and operability.

Chronic inflamatory processes starting from the vermiform may closely simulate malignant tumors and must be carefully distinguished.

Krokiewicz* alleges as of diagnostic value the absence of the diazo-reaction in intestinal carcinoma. This point awaits confirmation.

*Symptoms.*—As was stated at the beginning of this sketch, the symptoms of this disease are often elusive and vague, and the types show great variation. We may find a malignant tumor of the intestine with no symptoms at all, and we may find obstruction with no tumor. The best practical classification of the symptoms seems to me that of Boas, which I will slightly modify, placing in

Class I. those cases in which the subjective symptoms are so clear that no objective signs are really necessary, *i. e.*, those in which the history is sufficient for the diagnosis, the stenotic cases. Here we have a story for several months, say from three to eighteen, of obstinate constipation, relieved at first by cathartics, but later accompanied by attacks of intestinal indigestion, nausea, vomiting and colic, preceding a movement of the bowels, and passing off when the latter takes place. The interval between the attacks grows shorter as the constipation increases, until finally the attack becomes continuous, and we have the picture of complete obstruction. Nothing further need be said regarding the advisability of operating before the latter condition has supervened. The diagnosis ought to be made whether a tumor is present or not. An absolutely diagnostic point which is sometimes available is the gurgling of gas through

* Weiner Klin. Woch. 1898 No. 29.

the obstruction, and frequently the splashing of the liquid contents of the dilated and hypertrophied bowel above the tumor. Sometimes the contractions of the intestine above the tumor may be seen through the abdominal wall during an obstructive attack, and observed to disappear at the close of the attack with the relief of the obstruction, making a most valuable point for diagnosis and localization. With the temporary relief of the obstruction the symptoms may markedly improve and the patient gain in weight and color, conditions which may give rise to a false optimism as to the diagnosis.

More deceptive are slight cases, without visible or palpable dilatation of the bowel, where perhaps a single slightly distended coil may empty itself with a gurgle under the pressure of the physician's hand. This if repeated is a most important sign, and one that ought to be decisive. The reflex vomiting is important and may be mistaken for some gastric disorder. The vomitus is not characteristic and may contain hydrochloric acid, bile, etc. In contrast with vomiting from gastric catarrh, however, it may be observed to be unattended by fermentation, as evinced by the absence of the eructation of gas, and of sarcinæ.

The alternation of diarrhœa with constipation is frequent and generally is mentioned in all descriptions of the disease. As a matter of fact, diarrhœa may be on the one hand absent, and on the other hand may be present without constipation, as in the laterally situated, ulcerating, but non-obstructive growths. It is in these rarer cases that we are most apt to get blood, pus and mucus in the stools. This condition may lead to a wrong diagnosis of enteritis membranacea. The finding of small fragments of tumor in the stools is rare, but of course valuable. In

Class II. are placed cases in which the symptoms are of doubtful or non-explicit character, so that objective symptoms, such as the presence of a tumor, the finding of blood

or pus in the stools and the like, are necessary to diagnosis. In cases of this sort the value of exploratory laparotomy is great, and its danger so slight as not to be worthy of consideration.

In regard to presence of blood in the stools, it is a valuable point, but it occurs in so small a proportion of cases as to be often unavailable. Treves estimates it at 15 per cent. Blood and pus are both signs of ulcerating growths. The stools of small calibre, ribbon and lead-pencil stools, are rarely found, and that only in growths low down in the sigmoid, where they have not had an opportunity to become reformed and remolded after coming through the narrow portal of the obstruction. In

Class III. are placed cases in which local symptoms are entirely wanting until the disease has made extensive progress and perhaps metastasis has occured. Here a gradually increasing cachexia alone leads to a suspicion of disease of an internal organ. Such a case I have recently observed at the City Hospital. A man of fifty died with cachexia, ascites and jaundice from cancer of the liver, which presented an enormous tumor. In the right inguinal region during life could be felt a small nodule which was found at autopsy to be a cancer of the sigmoid, and the primary source of the liver cancer.

Class IV. includes cases in which sudden obstruction comes on without previous notable symptoms in the midst of apparent health. In these cases the existence of previous slight obstructive attacks may be brought out by careful questioning. Such cases as this serve to emphasize the fact again and again demonstrated in experience that abdominal symptoms apparently slight and transitory in their character may mean the insidious onset of fatal disease. The first case mentioned in this paper, perforation of a stercoral ulcer in a woman apparently previously healthy except for the disability incident to advanced age, is an illustration of this class.

In doubtful cases the general condition of the patient is often of value in diagnosis, but on the other hand may prove very deceptive. In some cases we find early a marked cachexia, in others no loss of weight, or at least a very slight one. In the stenotic cases we frequently find an alternating gain and loss. In the cases classed as ulcerative we commonly see a steady loss.

Now what are the dangers and benefits of operative treatment of cancer of the intestine? In the first place, in patients suffering from the stercoræmia resulting from long continued obstruction, a simple operation, even, such as colostomy, is fraught with grave dangers. In his collection of 426 cases, Bovis* found 101 cases of enterostomy with a mortality of 38.6 per cent., certainly a high mortality for so simple an operation as sewing the colon to the abdominal wall and opening it. On analysis we find that in a large proportion the operation was done late, with the patient *in extremis*, having had total obstruction for a week or ten days, and perhaps fæcal vomiting for a week. Of these grave forms of obstruction there were twenty-nine cases operated with twenty-four deaths, while of cases operated before obstruction had taken place, or just at its beginning, there were sixteen cases with only two deaths.

In regard to intestinal resection, or entero-anastomosis, the difficulties of technique and mortality are enormously increased if they are performed when the bowels are distended and paralyzed by complete obstruction. These operations ought never to be thought of under such conditions. The mortality from enterectomy for cancer in the 171 cases collected by Bovis was 31.5 per cent. Enterectomy performed under conditions of acute obstruction has a mortality of fifty per cent., nearly twice that of enterectomy in general.

Numerous cases are reported where a primary enter-

* Loc. cit.

ostomy has been successfully followed by a later excision of the growth which has been brought outside the abdomen at the first operation; and numerous ingenious methods have been devised to facilitate these procedures, which, however, we have no place for in a paper of this sort.

Now with regard to these operations for the removal of cancer of the intestine. What relief of symptoms and prolongation of life do they offer? The benefits need not be very great, for unoperated, the patient is face to face with certain death, not improbably under the painful symptoms of intestinal obstruction or peritonitis. Before attempting to consider the prolongation of life resulting from operation, we must first try to ascertain how long on the average a patient with cancer of the bowels lives before serious symptoms appear. The older writers assigned a duration of one to five months; König in 1890, followed by Salzer, Koch and Boas, assigned a duration of three to four years. As a matter of fact, the apparent duration which alone can be determined, varies enormously, but Bovis, who has made probably the most careful study of the question, assigns a duration of about one year before obstruction or peritonitis supervenes. This period is frequently much shorter, as evidenced in the cases of perforation and obstruction cited at the beginning of this paper as coming on in two and three months respectively. The shortness of the period of development is a weighty argument against delay, and in favor of early exploration. A delay of even a few weeks at a critical period in the growth of the disease may prove sufficient to allow it to get beyond control. Now supposing that we are dealing with a favorable case, *i. e.* one in which there are no metastases, and in which the tumor can be entirely removed. How long a survival will follow a successful enterectomy? Bovis gives a minimum average survival of 19.3 months. This is based entirely upon the time the patients were known to be alive,

so that there can be no doubt that if more of the patients had been followed during their entire lifetime the average would have been much higher. One of Boas' enterectomies was well five years after operation. Körte,* out of twelve excisions in 1900, reported five cases as well from three to eight and one-half years after operation.

Entero-anastomosis. This operation, being merely a palliative one, performed in cases when it is impracticable to extirpate the tumor, one cannot hope either for as long a survival or as great comfort as after enterectomy. Bovis gives a mean average survival of 6.4 months. This is undoubtedly below the actual average. Körte, in a series of nine recoveries from enterostomy, noted marked relief of symptoms in all cases. One patient worked for three years and lived three and a quarter years after the operation. Two patients lived more than a year without discomfort. Cases have been noted in which apparent decrease in the size of the cancer followed this operation. Dr. G. H. Monks recently reported such a case in which the patient lived in comfort for two years.

Enterostomy. For this palliative procedure Bovis gives a mean average survival of 10.1 months after operation, which, as in the case of the operations previously described, is undoubtedly too small. Senn reports a case of a survival of ten years after this operation, Lardennois a survival of five years. Fourteen of Körte's cases had a long survival after operation, one of them two years and seven months. In two of his cases life was prolonged three and one-quarter years and two years respectively by entero-anastomosis subsequent to enterostomy.

It is not an uncommon experience for most of us to see patients survive a year or more after left inguinal colostomy, and with a suitable truss to control the fæcal current, which

* Körte Verhand. d Deutsch. Gesellsch. für Chir. Berl. 1900. xxix. Th. 2.

can be arranged in most cases; the discomfort is not great.

Conclusions. To briefly review the operative indications and to state the answer to the question, In what cases of intestinal cancer, not emergencies, should operation be performed? I would say that operation is indicated absolutely in all cases in which a tumor is suspected of being a cancer of the intestine after careful eliminative diagnosis. If a benign tumor or obstruction is found so much the better.

Operation is indicated where the symptoms point to a probability of stenosis of the bowel whether a tumor is palpable or not.

Exploratory operation is indicated whenever vague intestinal symptoms associated with loss of weight in persons past middle life lead to the suspicion of intestinal cancer.

Exception should be made of cases of general metastases of the peritoneum, cancer of the liver, etc., in which event no radical operation can be proposed, but all operative measures must be directed to palliation.

With regard to the nature of the operation, that must be determined at the time in accordance with the nature of the individual case, and is not a question for elaboration in this paper. Suffice it to say that excision of the growth with immediate union of the ends of the bowel is the ideal to be sought. Intestinal anastomosis and enterostomy are merely palliative measures to be undertaken where the former is impracticable. The constant improvement which is being made in the technique of enterectomy promises an improvement in results.

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ARTICLE XLIV.

# OBSTRUCTIVE DISEASES OF THE LOWER BOWEL.

By HENRY O. MARCY, M.D.,
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The diseases of the pelvic viscera, which culminate in obstructive interference of the function of the lower bowel and bring into consideration surgical measures for relief or cure, constitute one of the most interesting chapters in the history of pelvic diseases.

The pathologic changes incident to such conditions are, for the most part, chronic, and are usually accompanied by discomfort, pain and suffering of an extreme character. The limitation of time at my disposal absolutely prevents an exhaustive treatment of the subject. For the sake of convenience, it may be well to sub-divide it,—first, the conditions extraneous to the bowel; second, the obstruction caused by its contents; and third, the pathologic conditions belonging to the viscus itself.

The changes incident to injuries of the pelvis, causing obstructive interference with the function of the lower bowel, are usually of an acute character and, as a consequent, do not come within the scope of the present discussion.

As the result of injuries, however, changes may supervene which materially impair the function of the lower intestine,

and, although not producing a positive obstruction, demand operative intervention.

Fracture of the coccyx is a well recognized illustration of such a condition. Deformation of the pelvis and osteoplastic growths also deserve mention.

In the male, diseases of the seminal vesicles and prostate frequently bring about such an irritated condition of the lower bowel as to induce most painful suffering and impair the function of defecation to a degree closely simulating intestinal obstruction. This is also true in old cases of stone in the bladder. Fortunately, owing to earlier surgical intervention, these conditions are far less common than formerly. Rarely chronic distention of the bladder may induce the most pronounced type of rectal irritation and be interpreted as a constant desire for defecation.

Incident to diseases of the reproductive organs in the female, interference of the function of the lower bowel to a degree demanding surgical intervention is by no means rare. The infectious diseases of the Fallopian tubes, with the secondary conditions dependent thereon, have, as a very common symptom, the disturbance of the intestinal function so as to produce the most acute suffering and, on this account, demand surgical intervention.

Small cystic tumors of the ovary or broad ligament not seldom bring about a train of functional disturbances of the lower bowel, seriously interfering with defecation, and occasionally by mechanical pressure make urgent demand for surgical relief. The retroversion of the gravid uterus sometimes produces the most marked tenesmus and straining, causing the patient, among other symptoms, an intense and unrelieved desire for defecation. Uterine tumors not seldom so block the pelvis as to arrest the function of the lower bowel, and for this reason render surgical intervention necessary. Unfortunately, this latter condition may come on so gradually as not to be suspected by the patient, and may be

easily overlooked by the physician. Instances of this type are by no means rare. I have operated upon a number of patients where the impairment of the intestinal function was the more prominent symptom because of a small uterine myoma having become firmly wedged in the lower pelvic strait. An illustrative case of this type is the following: Miss H——, aged 58. Menstruation ceased about ten years ago. Menorrhagia for some years was very pronounced. At forty she suffered severely from weight and distention caused by a large symmetrical fibroid tumor, distending the abdomen almost to that of full term of pregnancy. This slowly lessened in size after the menopause until it was believed by herself and her physician to have entirely disappeared. Six months previous to operation, vesical tenesmus and intestinal obstruction became so marked that she was obliged to take her bed and was too sick to leave it, her life being despaired of. I removed, with the greatest of difficulty, a calcified uterine tumor almost completely filling the pelvis and so firmly wedged in the lower strait that it required nearly my entire strength, aided by the Trendelenburg position, to dislodge it. Months followed before the intestinal function was completely restored.

The pathologic changes which occur in the hemorrhoidal plexus of vessels are sometimes so pronounced as to require surgical treatment, not alone because of the local conditions of pain and suffering, but that, co-associated with dilatation of the rectum, obstructive conditions of the lower bowel supervene. I have several times met with displacement of the pelvic viscera, causing an arrest of intestinal function to such a degree as to demand operation. Two cases of this character were incident upon hernia of the bladder, one in which for some days intestinal obstruction had been complete. The patient's condition was *in extremis* incident upon an old hernia in which eight or ten inches of the large intestine had long been incarcerated in the sac.

The medical practitioner is not seldom called upon to relieve acute distress from the accumulation of foreign material lodged in the lower canal. This is an experience common to all practitioners, and yet it is occasionally of a type and character demanding surgical intervention. Fecal accumulations are by far the most common, and the suffering incident thereto is often intense. This is more frequent in women, owing to the pathologic conditions of her pelvic viscera, dependent upon the result of the loss of perineal support, following child-birth, impairing the function of the lower bowel and inducing rectocele.

This condition is, however, not seldom met with in men and in women without other pelvic complications.

Some time since, I operated upon a woman with a history of more than thirty days having passed without defecation. The suffering was less than might have been supposed, and yet the entire lower bowel was blocked with such a mass of dried feces that, after complete etherization, the accumulated material was removed with much difficulty. Two cases have been reported to me in which the period of non-defecation was yet longer, and a considerable number of such cases are on record. It is very probable, however, that these conditions have been preceded by a long period of comparative functional inaction of the lower bowel, with previous distention, etc.

Foreign bodies are by no means rarely found interfering with defecation, demanding the intervention of the surgeon. It is remarkable that an individual may swallow accidentally such articles without knowledge thereof until arrested by the sphincter muscle. Pieces of bone are probably the more common. On one occasion the most acute distress demanding immediate relief was caused by a sharp thorn more than an inch in length, which I removed after it had penetrated the lower border of the sphincter muscle. Spasmodic contraction of the muscle was almost continuous, producing

the most intense suffering. The patient had no knowledge of its presence in the alimentary canal until thus caught in the act of defecation. Some time since a very intelligent lawyer brought me a round steel nail an inch and a half in length, bent at an angle of forty-five degrees. He was hastily eating in a railroad restaurant almost at the same time a hot biscuit and a baked apple. He felt a slight scraping in the throat, which he attributed to swallowing the stem of the apple. He had no knowledge of the nail until caught by the sphincter muscle in the act of defecation, producing intense suffering. Some years ago two immense gallstones were brought me for examination, having been lodged in the lower bowel from whence they were removed by the family physician. For months it had been believed that the patient was dying from cancer of the liver, so intense were her jaundice and the local suffering. Immediate relief was experienced and the patient began slowly to improve, when nearly six weeks later the removal of these gallstones, measuring over an inch in diameter, aided in making a more correct diagnosis. At the pathologic exhibit of the American Medical Association at St. Paul, in June, 1901, there was a calcareous concretion, so-called bowel stone, quite three inches in diameter, which had caused obstruction.

A most heterogenous collection of foreign materials are reported as having been removed from the rectum, many having been placed there by the sufferers themselves while not in stable mental condition.

The most important subdivision of the subject remains for consideration, viz., structural changes in the wall of the intestine, due to malignant growths. Since cancer of the intestine has been the subject of the two preceding essayists, it would be a fruitless repetition for me to enter into the discussion of the pathologic conditions except so far as relates to its surgical aspect. In a recent paper by E. N.

Mason,* entitled "Some remarks from an analysis of five thousand cases of malignant disease," the author draws some very interesting deductions. Sixty-three per cent. of the cases were females. This is shown to be due to the greater tendency of cancer to occur in the breast and in the uterus. The demonstration is made that by excluding all the cases of malignant disease occurring in organs peculiar to either sex, cancer is more common in the male, in the proportion of about fifty-three to forty-seven per cent. This the author traces more especially to trauma and syphilis. About three-fourths of all the cases of cancer in the male occur in the alimentary tract. His tables show that, in seven per cent. of the cases, the disease was located in the rectum, and that there were nearly twice as many males as females.

It is interesting to note that the committee, in discussing the predisposing causes of malignant disease, give the first place "to prolonged local irritation due to various causes, setting up inflamatory changes in the irritated tissues."

By far the greater emphasis is to be placed upon the early recognition of the malignant changes in the lower bowel, since in the treatment of cancer of the rectum little can be hoped for except from prompt surgical intervention before the disease has deeply invaded the tissues. I cannot emphasize too strongly the importance of a careful examination of the lower bowel in a large class of cases which are generally overlooked by the family physician. Unfortunately, the great majority of the sufferers from cancer of the rectum come to the surgeon when it is too late to hope for cure by complete removal of the diseased structures. When cancer has advanced to come within the strict limitation of my subject—obstructive disease of the lower bowel—little can be hoped for except palliation by colostomy. The usual place of selection is below the left anterior superior

* British Medical Journal, May 18, 1901.

iliac spine above Poupart's ligament. In this operation, it is usually the better way to make a funnel-shaped incision through the abdominal wall, parallel to Poupart's ligament, commencing just below the spine of the ileum. The sigmoid flexure of the intestine is easily found and is carefully sutured to the peritoneum, the needle being made to penetrate the connective tissue coat of the intestine. Good surgeons vary in the method of suturing. I much prefer fixation by two lines of fine continuous tendon sutures. Unless imperative, the intestine should not be opened until after twenty-four hours, when peritoneal union will be found to have taken place. The division of the bowel is best effected with the cautery knife. The mucous membrane may be drawn out and sutured to the skin, and the redundant rosette of the mucosa, which sometimes occurs, when this has not been done, is usually not painful or troublesome and aids materially in the retention of the feces.

The removal of malignant growths of the rectum is now advised in a very large percentage of cases not considered amenable to operation a few years ago.

The technic of these operations is well described in most of our modern text-books, and need not be repeated. In the reveiw of my own surgical experience I find that I have steadily enlarged the class of cases considered operable and am governed very much more by the evidence that the disease is absolutely local, that is, circumscribed within the periphery of the bowel at its original site of invasion, than by the precise location of the part involved. It is generally recognized that a circumscribed growth, limited to a portion of the bowel within reach of the finger, in either sex, is operable. If there is distinct evidence of glandular infiltration as a secondary development to the primary growth, it is very doubtful if operation is advised. This is the class of cases in which great relief is experienced and life prolonged by a colostomy, since thereby the part affected is given compara-

tive physiologic rest and can be kept from fecal defilement. We are indebted to Kocher for pointing out the very considerable increase of space obtained by the removal of the coccyx.

Little by little, portions of the sacrum were removed and a most noteworthy contribution was given, more especially by Kraske, after whom the operation is now named. In a number of instances, I have thought I obtained a decided advantage by first establishing an artificial anus, since by this means the pelvic structures are given a more nearly physiologic rest and can be operated on much more safely. One who has seen, for the first time, the operation for the removal of a considerable segment of the lower bowel performed by a free opening through the sacral region, especially in a moderately thin subject, is surprised at the wide field of operation thus obtained. The bowel may be encleated with the clear guidance of vision, and even the peritoneal cavity entered from below, freeing the intestine sufficiently to bring down a very considerable loop of the intestine. In this way I have several times resected a portion of the lower bowel, saving the normal anal aperture with the sphincter muscle.

In 1893 I first used a large Murphy button, for the coaptation of the segment, which came away the twelfth day. The thick intestinal wall rendered its application difficult, although the patient did very well. I have since had recourse to suture in addition.

This I believe to have been the first use of the Murphy button in this portion of the bowel, but its applicability was suggested to me by my friend Dr. H. O. Walker of Detroit. It will frequently be found impossible to save the distal end of the bowel, and then it will be necessary to free the upper portion sufficiently to bring the resected end easily out at the sacral portion of the wound and suture it there. If this has been done aseptically I think it preferable to close the entire

lower segment with lines of buried sutures, thus evenly coaptating the soft parts.

Although the wound is a large one the vascularization is ample and the union of the aseptic wound is primal. Special care should be taken in closing the skin with a light-running buried suture which should include only in the deeper portion of it, since otherwise the exceptionally large follicles of the skin may prove a source of infection. This portion of the wound is carefully sealed with iodoform collodion.

Another advantage of this operation, by a wide invasion of the pelvis from below, lies in the possible removal of many of the pelvic glands, a common source of secondary infection. The artificial anus in the sacral region is quite as troublesome as in the iliac region. The mortality following these operations is much less large than even a decade ago, while the results are much more satisfactory. First, because of a better aseptic technic; and secondly, because the cases operated upon are brought to the surgeon as soon as the diagnosis has been made without the fatal delay of tentative treatment which prevailed at an earlier period. The future of this class of operations gives yet better promise, since many details of the surgical technic come within the limit of great possible improvement. It is very probable also that a further specialization of this regional surgery will obtain, since the severer operations are likely to tax the resources of the best surgeons, and a greater familiarity with the local technic aids materially in securing an effective result.

Probably the most valuable contribution to the surgery of the lower bowel is due to the teaching of the gynesic surgeon. The invasion of the abdominal cavity and life-saving results dependent thereon form one of the most brilliant chapters of modern science.

The opening of the abdomen with the patient in the Trendelenburg position places the entire pelvic viscera under

easy inspection. The small intestines are walled off from above, the peritoneum is opened from below, and the lower portion of the bowel is freed from its attachments. Usually one or two branches of the sacral arteries are divided and require ligature. Then the loop of the intestine is freed and the diseased portion treated as the conditions demand. If the lower segment is free from disease sufficiently large to permit of its utilization it should be preserved. If not, it must be removed. Then it will be necessary to doubly ligate the upper portion of the intestine and divide between the ligatures. The upper portion is cleansed and carefully protected from infecting the parts, while the lower segment is entirely removed. This having been done, the bleeding vessels are ligatured and an artificial anus is made by securing the upper healthy bowel laterally as in a usual colostomy, and the entire pelvic wound is closed with buried sutures, much care being exercised in closing the peritoneum of the pelvic basin. If it has been possible to maintain an aseptic technic it is better to close the pelvic wound without drainage; otherwise drain. If the upper loop of intestine is of sufficient length it may be wise to make the artificial anus in the normal site, possibly saving some of the sphincter muscle.

In woman the vagina may be utilized to "piece out" the abbreviated bowel. When this is done much care must be used in closing the pelvic peritoneum about the displaced intestine. When it is possible to resect the diseased portion of the bowel, the intestine is freed as above described, inclosed by ligatures and resected. Reunion of the healthy portions may be effected by retention apparatus, as the Murphy button already referred to, or by sutures. It must be remembered in either case that the connective tissue of the intestine is the important portion for coaptation. Dr. John B. Wyeth of New York, at the present June meeting of the American Medical Association, described a modifica-

tion of the combined operation which he had devised and used in three instances.

The sphincter is well dilated and, as far as may be, the lower bowel is emptied and disinfected. The abdomen is entered from above, the peritoneal attachment of the pelvic floor freed, the bowel separated and the circumscribed portion of the diseased structures tied off and removed as already described. Through the lumen of the lower segment of healthy bowel long forceps are passed and the upper portion of the freed bowel drawn down externally. This also everts the lower segment and makes easy the careful suturing of the two portions of the bowel. After this has been effected, the everted bowel is restored, the bleeding vessels are sutured, the peritoneum of the pelvis is carefully approximated, and the abdominal wound is closed. The perineal wound is drained.

I regard the resection of the lower bowel for cancer by approaching it from above in many instances as a very great advance in modern technic for a variety of reasons. The lymphatic glands of the pelvis can be examined and removed if necessary as by no other route. The resection may be made much more accurately and in many instances the function of the lower bowel preserved or restored. Cancer of the rectum is one of the most deplorable of all diseases. The last decade has, however, added greatly to the improved surgical measures for its relief and cure, but no field of surgery demands greater improvement in skill and technic or promises greater triumph in the relief of suffering.

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## DISCUSSION.

Dr. W. N. Swift, of New Bedford: I have seen a number of cases in patients over sixty years of age, where there has been a history of digestive disturbances extending

over six months to more than a year, terminating finally with symptoms of obstruction, coming on rather gradually. In my experience vomiting has not been prominent. The general health of the patients has been somewhat affected, but not in a marked degree, and in none of these cases has there been what could be called a cachectic condition. With one exception the patients have had malignant disease of the sigmoid flexure or descending colon. The point I wish to emphasize is, that these cases do not usually show marked symptoms of malnutrition, and that malignant disease is not made improbable by the absence of so-called cachexia.

Lately I operated on a man of sixty with symptoms of chronic intestinal obstruction, and found malignant disease of the upper part of the sigmoid flexure too extensive for excision. I did an iliac colotomy, separating the fibres of the abdominal muscles without cutting any. The result has been satisfactory, as the patient has good control of the discharges. Abdominal distention, I believe, is a very important symptom in these cases, and when it occurs with obstipation is an indication for abdominal section.

Two years ago I had a case of a woman of 60 years. She had complete intestinal obstruction, that had come on gradually. When I saw her she had extreme abdominal distention, and her condition was such that no operation was justifiable. She died the next day. The autopsy showed a stricture at the point of junction of the colon and sigmoid flexure. There was a dense mass of cicatricial tissue and complete obstruction of the intestine at that point. The cause of the formation of this cicatrix was not made clear by the history. In cases of chronic intestinal obstruction, an exploratory abdominal section should always be made; if the disease is not malignant a radical operation can be done, and if it proves to be cancer too extensive for excision, an artificial anus gives the patient much relief.

A few weeks ago a negro of fifty-five was brought to the hospital. Seven days before he had been taken with abdominal pain. His bowels had been constipated. His pain began in the evening and was not severe enough to keep him at home. The next day the pain increased, was spasmodic in character. At night he proposed to go to a

meeting where he was to wear a uniform. He attempted to put on his belt and found he could not do so. The pain and abdominal distention continued to increase. He was given castor oil and other cathartics, which increased the pain, but did not move the bowels. He did not vomit. He had no rise in temperature. He was given large rectal injections. The day that he came to the hospital he was given about a gallon of water, which he retained.

On section I found a loop of intestine running diagonally across the abdominal cavity from the region of the cecum to high up under the diaphragm on the left side. It was enormously distended and filled the abdominal cavity. After emptying it I found it to be the sigmoid flexure with a complete twist in its mesentery. The small intestine was completely collapsed. The patient stood the operation quite well, but was in no condition for excision of the intestine, and died in twelve hours.

This is an example of a case where much precious time was wasted. Cathartics in these cases do harm, and injections only torture the patient and do no good. In this case the twist allowed the injected fluid to enter the loop, but it only increased the trouble. I think whenever an intestinal obstruction is suspected surgical interference is indicated. The reason the mortality of intestinal obstruction is so high is on account of senseless waiting.

ARTICLE XLV.

# UNDER WHAT CIRCUMSTANCES (EXCEPTING EMERGENCIES) IS IT DESIRABLE TO OPERATE UPON GALL-STONES FOR RADICAL CURE OR FOR RELIEF.

By MAURICE H. RICHARDSON, M. D.
OF BOSTON.

A CONSIDERATION of the surgery of gall-stones is practically a consideration of operations for radical cure. Operations for relief are to be considered in those rare instances in which, as the result of prolonged ulceration in the common duct, a stricture is formed after expulsion of the stone —causing permanent jaundice. Such a lesion can be remedied only by cholecystenterostomy, or possibly by providing a new outlet for the duct above the stricture into the duodenum. And yet this rare lesion is not to be discussed in connection with the palliative treatment of gall-stones, for the gall-stone has escaped. It is one of the remote ill effects which gall-stones occasionally produce, and for the prevention of which early operations upon gall-stones are desirable.

The removal of gall-stones as a palliative measure may be demanded in hopeless diseases associated with gall-stones, —cases in which the suffering is dependent largely upon the gall-stones themselves, or is at least aggravated by

them. Removal of such gall-stones is radical with reference to the gall-stones, but palliative with reference to the hopeless disease. In a strict sense no palliative operations are possible upon gall-stones themselves; they either are removed or they are not. The scope of this paper, however, does not include a discussion of the palliation of malignant or otherwise hopeless disease by the removal of pain-causing gall-stones. Suffice it to say, that under some circumstances such a procedure may be justifiable.

The proposition to be discussed in the following pages is as follows:—

*Gall-stones should be removed at the earliest favorable moment after the diagnosis has been made, unless there are contra-indications in other viscera or in the patient's general condition.*

Inasmuch as the diagnosis of gall-stones can be made only when they begin to offend (excepting of course during abdominal operations for other causes), gall-stones should be removed either as soon as they begin to offend or at the most favorable period after their immediate ill effects have had time to subside. By *the earliest favorable moment* I mean that period of time when there is no infection of the gall-bladder to contaminate the field, no impaction in the common duct to increase the difficulties and dangers of dissection, and no jaundice to induce hemorrhage or to impair the patient's power of recovery. In many cases the favorable moment follows recovery from the disturbances of a transitory biliary colic unattended by jaundice; in others it follows the disappearance of jaundice after passage of the stone; in others, the subsidence of fever and other signs of a biliary infection. In all cases of jaundice one should wait a reasonable time for that favorable moment, in the hope that the stone may escape from the common duct into the duodenum, and that bile may reappear in the stools.

The arguments in favor of this proposition are :—

(1) The operation is as a rule easy and safe, and all stones are quickly removed.

(2) The remote dangers of gall-stones are either avoided or lessened. These are serious disabilities, grave emergencies and malignant disease.

(3) If the diagnosis of gall-stones proves to be wrong, other lesions may be discovered and remedied,—lesions perhaps even more serious than those of gall-stones.

(4) Late operations upon gall-stones are, as a rule, difficult and dangerous. Operations made imperative by progressive and lethal symptoms must be performed under great disadvantages and dangers: the gall-stones are generally more inaccessible, the dissections deeper, and the patient's powers of resistance lessened.

Against the proposition are the following arguments :—

(1) There is some danger in the operation, though it is but slight.

(2) The diagnosis may be wrong and the exploration unnecessary.

(3) There is the possibility of hernia in the scar.

(4) There is the possibility that gall-stones may recur.

(5) There is the possibility of spontaneous cure.

(6) There is also the possibility that, after offending enough to prove the diagnosis, gall-stones may give no further trouble.

(7) The last and decisive attack of biliary colic may have been caused by the last remaining gall-stone, exploration showing that none remain.

Unfortunately the manifestations of gall-stones are extremely varied. We cannot assume that the early-diagnosticated cases are of the simple, easily accessible and safely operable kind, and the late of the inaccessible and dangerous kind; for the very earliest manifestations of gall-stones may place some of the cases immediately in the

dangerous category, whereas the latest may place them in the simplest and most easily operable variety. The arguments of simplicity and safety in operations performed as soon as the diagnosis is made will therefore not apply in cases of common duct impaction, for the operation will not be either easy or safe. Moreover, the late operations will not be necessarily hazardous in all cases, for the gall-stones may be confined to an easily accessible gall-bladder. Presumably, however, gall-stones when they begin to offend are confined to a normal or comparatively normal gall-bladder in a patient of good general condition, while later in their history they will be deeply situated in patients of poor general condition.

(1) Gall-stones should be removed at the earliest possible moment after the diagnosis is made, in the absence of contra-indications, because the operation is as a rule easy and safe, rapid and effectual.

This argument needs no amplification to those familiar with the surgery of gall-stones. To those who are not familiar with the operative treatment of gall-stones, it should be said that when the gall-stones are confined to the normal gall-bladder, it is always possible to bring the gall-bladder to the wound, and to remove the gall-stones without contamination of the field, except perhaps to a very slight degree. If bile does escape into the field of operation, that bile is usually sterile, and does no harm. The gall-bladder can be stitched to the abdominal wound and it can be drained as long as desirable. The mortality is so small in healthy individuals that it need hardly be considered. The danger is less than that of the passage of a single stone from the gall-bladder to the duodenum, and it is less than the danger, constantly present, of infections of the gall-bladder through the irritation of the gall-stones themselves. Robson has recently stated that he has never lost a case of cholecystotomy for gall-stones undertaken under these cir-

cumstances. Though my experience is much smaller than Robson's—probably not over a hundred operations that belong to this category—I have had in simple cholecystotomies no deaths whatever. It is safe to say that such is the experience of most operators.

In the great majority of cases the diagnosis of gall-stones depends upon repeated attacks of biliary colic, the history often embracing many years. The application of common-sense principles of surgery is fortunately diminishing the number of cases of prolonged and repeated gall-stone attacks: patients now do not wait until they are compelled to submit to operation by unbearable pain and ever-increasing disability. Still, in far too many cases the favorable moment is lost by waiting through many recurring attacks of pain for the diagnosis of gall-stones to be made. When operation is undertaken after a few attacks, and when there have been no periods of prolonged jaundice; when, if there has been jaundice, it has entirely disappeared at the time of operation, the gall-stones, if any remain, will be found in the gall-bladder, and the case will probably belong to the category of the safe, easy and successful operations. When the very first attack of biliary colic is quickly followed by a permanent jaundice, if the diagnosis is at once made and the patient at once operated upon, the prognosis is still favorable, because the great changes wrought in the gall-bladder and the contiguous viscera by prolonged cholelithiasis will not have taken place. The gall-bladder will be but little changed; its walls will be elastic, distensible, easily attached to the abdominal wound; and there will be no adhesions about it. Dissection and isolation of the common duct will be easy; crushing of the stone in the duct between the thumb and finger will often be feasible because of the presumable softness of a recently formed stone.

In some rare instances a stone of great size and of great age that has lain innocuous, perhaps for many years, will

give the first signs of its presence by obstruction to the biliary flow. Jaundice—perhaps without pain, or with merely a feeling of distress—will be the only symptom upon which to base an opinion. The diagnosis in such cases is always doubtful. Often the surgeon will find malignant disease of the pancreas, cancer of the gall-bladder, or some other hopeless condition. The argument of safety, feasibility and success will not apply to such a case; the operation may be of the greatest difficulty, the greatest magnitude, and the greatest danger. Still, great as the difficulties and dangers of this operation must be, they will be less if the operation is performed as soon as the diagnosis is made than if it is performed at a later time.

(2) Because the remote dangers of gall-stones are either avoided or lessened.

The remote dangers of gall-stones are not sufficiently appreciated. Many of those who oppose the surgical treatment of gall-stones as a routine measure do so because gall-stones which have given no sign of their presence are so frequently found at the autopsy table. I doubt if those who rely upon the records of autopsies for this argument appreciate that other argument, demonstrated so frequently upon the operating table, that the remote effects of gall-stones may be—and frequently are—of the most serious and lethal nature. I have not often seen these things demonstrated post mortem; but I have so many times demonstrated them myself at operations that I am deeply impressed by their significance. When gall-stones begin to offend, in many instances they do so by infection of the biliary passages. The proportion of cases of infection it is impossible to give, but I have no doubt that it is a large one. The infection is not necessarily a serious one: it doubtless often exists without marked symptoms. Gall-stone attacks, accompanied by fever and tenderness in the gall-bladder, are in reality infections of the gall-bladder. These infections

result, in some cases, in an acute cholecystitis of the most serious character, often ending in abscess of the gall-bladder or in gangrene and rupture. In the great majority of cases, however, it results in a sub-acute inflammation, with thickening of the gall-bladder walls, and a peritonitis with adhesions to contiguous viscera. In many cases the thickened and friable gall-bladder is found contracted upon gall-stones, and contains also highly infectious fluids. In such cases it is impossible to bring the gall-bladder to the abdominal wound; moreover, the walls are so friable that they do not permit suture. Removal of a stone from such a gall-bladder makes the field septic, for the gall-bladder contents are septic. The dangers of peritonitis are much increased; the dangers of hemorrhage are much increased, especially if the patient is jaundiced; the danger of shock and exhaustion are much increased. The blood vessels are friable; the mucous membrane bleeds freely; the bleeding may be hard to check,—it may even be fatal, especially if the patient is jaundiced. Plastic operations upon the gall-bladder are impossible. Adhesions to the stomach, to the pylorus, to the duodenum, to the liver, to the transverse colon, are sometimes so firm that isolation of the gall-bladder and of the common duct is extremely difficult and bloody.

A not uncommon remote effect of this condition is malignant disease. I have seen—time and again—a malignant tumor filling the gall-bladder and confined apparently to it. I have seen the liver a mass of malignant disease starting presumably in luxuriant new growths bursting from the gall-bladder itself.

In many instances the gall-bladder shows evidences of having discharged a stone by direct ulceration through its walls into the duodenum, with subsequent spontaneous cure. In many the ulceration leads directly into the peritoneal cavity and causes fatal peritonitis: in some it re-

sults, through adhesions, in abscess of the abdominal wall and expulsion of the stone.

Remote changes in the common duct, in the hepatic duct, in the liver, dependent upon gall-stones, are too frequent to need comment. Infectious processes, if they do not invade the gall-bladder, may extend into the radicles of the hepatic duct. In impactions of the common duct, the hepatic and common ducts above the impaction are dilated and thickened, and the walls friable. The gentlest manipulations sometimes tear them, so that suture is difficult if not impossible.

Finally, owing to gall-stones, there are those changes in the pancreas, the nature of which we know as yet so little, but which recent investigations seem to prove of the utmost importance,—acute and chronic pancreatitis, pancreatic hemorrhage, and fat necrosis.

A condition of the most serious nature directly dependent upon gall-stones we see in the constitutional effects of jaundice. When, as the result of impactions in the common or in the hepatic duct, the patient has suffered for many months from profound jaundice, the operation will present unusual difficulties; removal of the stone will require the deep, extensive and hazardous dissections of choledochotomy; incision into the duct will endanger the portal vein and other structures; repair of the incised duct will demand great patience and manual dexterity; and, finally, these dangers will be imposed upon a patient so depressed by prolonged jaundice as not easily to withstand them. Worst of all, the surgeon may be confronted by that grave complication of surgery—hemorrhage,—and possibly a hemorrhage which he will be unable to control.

To illustrate some of the foregoing statements by recent examples—I recall the death of a strong man of middle age within the past year from uncontrollable capillary hemorrhage after the removal of a stone from the common duct.

Years ago I drained the gall-bladder for acute infection. In 1900, I removed a stone from the cystic duct without detecting anything in the common duct. A persistent biliary fistula, with absence of bile from the stools, proved an obstruction of the common duct,—where, in 1901, I found the stone and removed it. Not a single spurting artery was met with in the dissection. As the operation progressed, however, every capillary began to bleed. As fast as the bleeding points were tied, others appeared. The man died in the course of 24 hours, of capillary oozing, which we were unable to check.

Within the last six weeks I have operated for gall-stones, and have found them associated with cancer of the gall-bladder. The operation was followed soon by a disseminated carcinomatosis which proved fatal. In another case six stones were removed from the common duct (operation in Springfield on June 5th), and three large stones from a contracted gall-bladder. Another was crushed in the common duct between the fingers. This patient died of exhaustion on the third day. In June (in Newton) I removed an apparently malignant gall-bladder which contained a single gall-stone and a dark lardaceous substance resembling inspissated pus mixed with fat. Dr. Whitney reported the gall-bladder non-malignant. This patient has made a splendid convalescence.

In all these cases there had been long-standing symptoms of gall-stones; there had been repeated attacks of pain. The gall-bladders were contracted, thickened, friable; the parts were glued together, and everything was obscure. The operations were of the greatest difficulty. There was no chance whatever of spontaneous cure. If these patients had been operated upon early, the conditions found at the time of operation would have been very different, and the operations undoubtedly successful. This is the kind of operation to which the palliative treatment of gall-stones drives

the patient. It is in this formidable class that the danger and the mortality lie. Unfortunately, the responsibility for failure is placed too often at the surgeon's door,—where it surely does not belong.

(3) If the diagnosis of gall-stones proves to be wrong, other lesions may be discovered and remedied,—lesions perhaps even more serious than those of gall-stones.

The strength of this argument lies in the difficulties of exact diagnosis when the symptoms point to the epigastrium and right upper quadrant. The diagnosis of gall-stones is in typical cases easy,—dependent upon a history of repeated biliary colics, with or without jaundice. The diagnosis, however, even in cases which are apparently the plainest, may be mistaken, unless, after a transitory colic, one or more gall-stones are found in the stools. Even when the diagnosis of gall-stones is apparently proved by the discovery of them in the stools, there may exist other lesions as serious as that of the gall-stone itself,—or even more serious:—acute or chronic pancreatitis, gastric ulcer, duodenal ulcer, obstructions at the pylorus, new growths, faulty adhesions.

The wider a man's experience in actually exploring the regions of the gall-bladder and pylorus, the more he is impressed by the difficulties of exact discrimination between lesions in this locality causing pain. If then it is maintained that explorations should be made as soon as the diagnosis of gall-stones is made, it becomes important to discuss those lesions which may be found in case the diagnosis of gall-stones is wrong. If under that mistaken diagnosis exploration should prove invariably harmful or even useless, the possibility of such mistake would be, in doubtful cases, a strong argument against exploration. Fortunately some good will be accomplished by the exploration, even if the supposed cause—gall-stones—is not present, for there are few causes of pain simulating gall-stones strongly enough

to justify a diagnosis which in themselves do not sooner or later demand operation. Many of them require operation at the earliest possible moment, if operation is to be of any real benefit. To this category belong, for example, tumors of the pylorus, of the gall-bladder, of the intestine, and even of the kidney. In my last service at the Massachusetts General Hospital I explored in one case the region of the gall-bladder for frequent attacks of intense pain. No lesion whatever was found, except a flattened tumor of the anterior wall of the pylorus. This tumor, which I removed by resection of the pylorus, proved to be a beginning carcinoma. It was, as I say, at its very beginning, and was a flattened, warty growth which suggested the keratosis of the back of the hand seen years before the characteristic appearances of epithelial cancer have shown themselves. The exploration in this case gave the young woman—she was only twenty-one—the very best chance to escape the horrors of pyloric cancer. The diagnosis of gall-stones was never positive; but it led the surgeon directly to intervention at the most favorable moment in pyloric cancer.

In a patient with supposed tumor of the gall-bladder dependent upon gall-stones I found a pyonephrosis with completely disorganized kidney. Operation on the kidney was brilliantly successful, and the patient remains well. In several instances exploration has shown constriction of the pylorus.

I have never met with duodenal ulcer or other disease of the duodenum suggesting gall-stones; but there is always this possibility to be borne in mind.

Early explorations will occasionally show a tumor of the gall-bladder itself admitting of extirpation, or a tumor of the liver close to the gall-bladder. In one instance I found, on exploring what was supposed to be a diseased gall-bladder, a carcinoma of the hepatic flexure of the colon, and this at a stage when extirpation was quite feasible.

More important perhaps than any other lesion suggesting gall-stones, and relieved by operation, are the chronic inflammations of the pancreas. I have found in a number of patients what seemed to be a tumor of the pancreas associated with a jaundice supposed to be the result either of gall-stones or of pancreatic cancer. A hard, irregular tumor at the head of the pancreas, perceptible by palpation after opening the abdomen—especially if the history has been one of jaundice and emaciation without pain—has always seemed sufficient evidence of pancreatic cancer to justify abandonment of the operation. The experiences of the past year have led me to suspect that, in some of these cases at least, the evidence obtained by palpation is insufficient to establish the diagnosis of cancer; that jaundice and its accompanying symptoms may be due to the impaction of a stone in that part of the common duct surrounded by the head of the pancreas, and that the stone there impacted may cause a sensation of irregularity and resistance very strongly suggestive of malignant disease. I fear that I may have left the patient unrelieved in several of these cases. In a recent one (June 5th) at Springfield, a patient of Dr. Chapin's, after removing several stones from the gall-bladder, and six from the common and hepatic ducts, I felt in the head of the pancreas a hard and irregular tumor that seemed to be a nodule of cancer. I was determined to be sure, however, and found after repeated manipulations, that this tumor was caused by the impaction of several gall-stones in the pancreatic portion of the common duct. One of these I squeezed back and out of the incision; the others I crushed between my thumb and fingers into a mass of fine detritus, and delivered in the same manner. After this procedure the head of the pancreas felt perfectly normal. This patient died of exhaustion on the third day.

In another case, operated upon in Fall River, in May, 1901, I felt a similar tumor in the head of the pancreas.

This hard mass resolved itself into a doughy one after manipulations with the thumb and fore-finger, and demonstrated the probability of an impacted stone at that point. Renewing a few moments later the process of crushing, I could feel nothing whatever. This fact led me to believe that the detritus had all passed into the duodenum. This patient is now well.

In another case, already published in the Philadelphia Medical Journal of October 6, 1900, I found on exploration a large tumor of the head of the pancreas. The symptoms had been pain and transitory jaundice, and there was an ill-defined resistance near the usual situation of the gall-bladder. The diagnosis was gall-stones, but no suspicion of involvement of the pancreas was entertained. When exposed, the suspected gall-bladder proved to be an enlarged and resistant pancreas. The tumor, which involved chiefly the head of the pancreas, was supposed to be carcinoma and inoperable, though no actual demonstration of the tumor was made other than by digital exploration. A few small gall-stones were removed from the gall-bladder, and drainage was established. This operation was performed before the publication of Robson's article on the subject of chronic pancreatitis, and I was therefore much gratified, when discussing the subject with him shortly afterward in London, to be assured that there was a possibility in the case of a permanent recovery after temporary drainage of the gall-bladder. The patient has made a brilliant and thus far permanent recovery.

I could bring forward many other cases tending to prove the truth of my proposition. It seems, however, needless to go further in establishing what experience and common sense show: that the practical application of this proposition would mean the safest and most efficacious remedy at the earliest possible moment, not only for gall-stones and for their immediate and remote effects, but for tumors of the

pylorus, benign and cancerous; for extensive and faulty adhesions of the parts about the gall-bladder, the liver, the stomach, the pylorus, the duodenum, the colon; for ulcers and other affections of the duodenum; for new growths of the gall-bladder itself; for tumors of the liver simulating tumors of the gall-bladder; for tumors—benign and cancerous—of the hepatic flexure of the colon; for acute and chronic diseases of the pancreas suggesting gall-stones; for tumors of the kidney simulating tumors of the gall-bladder. With the exception of diseases of the duodenum, I have found and, in cases not essentially hopeless and not too far advanced, have been able to remedy, all these lesions, when the diagnosis has been perhaps as much in favor of gall-stones as of the exact disease found.

(4) Late operations upon gall-stones are difficult and dangerous.

Every one experienced in the surgery of gall-stones will admit, I think, that the mortality in gall-bladder surgery is almost wholly confined to those cases in which intervention is a late or a last resort. Many physicians, however, who perhaps have not seen much of the surgical treatment of gall-stones in their early stages have been led to believe that the large mortality is the result of the operation rather than the result of delay. Such must be reminded, again and again, that all surgical measures—no matter how trivial in themselves—may be extremely dangerous when made imperative by certain conditions dependent upon gall-stones.

The common source of increased danger is jaundice, which enfeebles the patient, induces hemorrhage, and in other ways increases the risk and the mortality of operations. In prolonged jaundice the patient may succumb, in the natural evolution of the disease, to hemorrhage from the mucous membranes, to secondary disease of the kidneys, or to acute affections which in a state of health the patient could easily withstand. If operated upon, he may die from

excessive and uncontrollable capillary hemorrhages. A second source of increased danger is the extensive dissections necessary for the removal of gall-stones from the deep biliary passages. Furthermore, these dissections must be made during the systematic depression of more or less prolonged jaundice, as a rule, in close contiguity with the portal vein, the inferior cava, the pylorus, and the duodenum. They are especially hazardous if the long-continued presence of gall-stones has resulted in the formation of firm and dense adhesions. In a word, the operations are in themselves more difficult and extensive, the patient's power of resistance diminished and the mortality therefore increased.

On the other hand, when the hepatic and common ducts are free, late operations upon the comparatively normal gall-bladder may be as easy and as safe as operations performed as soon as the diagnosis is made. When, however, as the result of prol ɔnged gall-stone irritations, the gall-bladder is contracted and friable, when its walls are thickened and adherent, when its contents are infected and purulent, the dangers are great, not only from the increased technical difficulties, but from the liability to wound infection and to peritonitis.

In many instances it may be necessary to remove the gall-bladder entirely. This operation, though usually successful, is often difficult, and may be fatal. In one of my cases death followed total extirpation of a contracted and thickened gall-bladder. Even if extirpation is not indicated, the contracted gall-bladder must be drained, after removal of its stones. Removal of gall-stones from a long-inflamed gall-bladder is not always easy. I have found hour-glass contractions requiring lateral and difficult incisions, stones encapsulated in ulcerated and thin-walled diverticula, stones impacted deep in the cystic duct, abnormalities of position in the gall-bladder itself, with adhesions so dense and thick that dissections were necessarily blind and hazardous. In

one instance attempts to remove an impacted stone through a contracted gall-bladder resulted in the escape of the stone through the walls of its nidus into the depths of the subphrenic space, whence it was removed with the greatest difficulty,—an operation of the most formidable kind and one which proved fatal. The physician who sent me this patient had previously referred to me an old lady with jaundice. Her gall-bladder was empty, and a permanent biliary fistula followed cholecystotomy. She had some obstruction in the common duct which the operation failed to relieve, and she never quite forgave the surgeon, though the operation, by relieving the jaundice, even at the expense of a biliary fistula, prolonged her life. Based upon these two cases a physician's experience might well make him skeptical about late gall-bladder surgery, though it should be a potent argument in favor of early. Even, however, in the late operations upon the inflamed and contracted gall-bladder the success is, as a rule, great. The analogy between operations upon the gall-bladder and operations upon the vermiform appendix is striking. Could every appendix be removed at the time when it is possible to close the abdomen without drainage, there would be practically no mortality in appendicitis. Could every gall-stone be removed when it is contained in a normal gall-bladder, there would be practically no mortality in gall-stones. The moment, however, that the infection breaks through the appendix, the fatal element of sepsis comes in. So in gall-bladder surgery the moment the gall-bladder becomes itself infected, or as soon as the gall-stone—impacted in the common duct—obstructs the biliary flow, the grave dangers of sepis or of jaundice appear. No other argument than the argument of increased danger is really necessary to demonstrate the truth of my proposition. But, as will be discussed later, many patients with gall-stones recover spontaneously,—or apparently recover,—and the physician who sees patients

recover after transitory colics may be led to expect them to recover after the jaundice of permanent impactions. Instead of condemning that palliative course which has made a late, dangerous, and perhaps fatal operation imperative, they condemn as unwarranted that operation which gives the only reasonable hope of recovery.

I recall the remark of a physician who had allowed a patient to drift through months of hopeless jaundice—with all that that condition implies—for the relief of which a delayed operation proved fatal from hemorrhage: "I shall never forgive myself for allowing this operation." Those of us who heard the remark thought that it would have been more appropriate if he had said: "I shall never forgive myself for not having advised this operation earlier."

The argument based upon the great dangers and mortality of late operations would need no such amplification as has here been given were it not for the belief, more or less prevalent, that medical treatment, which—in my opinion at least—can have no effect whatever upon the gall-stones, does in fact cure them, and that there is enough expectation of spontaneous cure to justify the immediate if not the remote dangers of delay.

That medical treatment has the least beneficial effect in aiding the mechanical expulsion of a stone through the biliary passages from the gall-bladder to the duodenum, either in diminishing the size of the stone, in increasing the lumen of the passage, or in increasing the force of expulsion, seems to me absurd. To allow the patient to undergo, once or twice, the slight dangers of a biliary colic, in the hope that the last stone has been passed, or for the establishment of the diagnosis, seems reasonable enough, for the dangers are not great. Small as they are, however, they are greater than the dangers of an early cholecystotomy. To persist in palliation when there has been the least sign of a gall-bladder infection, of a common duct impaction

(jaundice); or when, from the discovery of facetted stones in the stools there is good reason to suppose that more remain, is to restrain the patient from the course of safety, and to force him into the course of danger. It is against reason, experience, and common sense.

The lesions of gall-stones are mechanical; their treatment should be mechanical.

Against the foregoing proposition are to be considered the following arguments, some of which have already received brief mention:—

(1) There is some danger in the operation, though it is but slight.

It is well always to emphasize this argument; and it is, I think, safe to say that the greater a man's experience, the more he appreciates its force. The risks in operations for gall-stones undertaken at the most favorable time are the risks of anæsthesia, the risks of simply opening the abdomen, and finally, the risks of opening the gall-bladder itself. The risk of opening the abdomen under our present methods of asepsis is slight, but still it exists. The risk comprises unperceived errors in asepsis; inadequate preparation of the field; errors in the sterilization of the instruments, gauzes, linen, and especially of the hands; if gloves are worn, the risk includes imperfect sterilization of the glove, the tearing or pricking of the glove, and the escape into the wound of fluids that during the operation have been macerating the fingers; if no face mask is used, it includes the dropping of perspiration and of hairs into the field,—the coughing, sneezing, or talking of particles of buccal or of nasal fluids into the wound. There are the accidents of technique,—the cutting or tearing of the intestine, the possible contamination of the wound with infected bile; the dangers from the operator's technical treatment of the gall-bladder. There are also the dangers depending upon the

idiosyncracies of the patient: his inability to coagulate his blood,—perhaps an inherited tendency to hemorrhage.

Taking all these dangers together, they do not, in my opinion equal the dangers attending the passage of a single stone from the gall-bladder to the duodenum.

The scope of this paper does not permit a discussion of methods of asepsis, as it is taken for granted that he who is to operate for gall-stones is a surgeon of experience who uses only most thorough methods of asepsis. If the man who proposes to treat gall-stones surgically has had no experience in abdominal surgery, or such limited experience as practice in a narrow field affords,—and especially if he has not had the opportunity of assisting in a large number of cases,—the dangers of the operation will, in my judgment, exceed the dangers of palliative treatment, and the operation should not be undertaken. Operations upon the gall-bladder for gall-stones—like most operations upon the abdominal viscera—may suddenly demand resources that only large experience and great skill affords: the operator must be prepared to meet, in what are apparently the simplest cases, the gravest emergencies of abdominal surgery.

(2) The diagnosis may be wrong and the exploration unnecessary.

This topic has been considered under a previous argument. The diagnosis is frequently wrong. The exploration will have been proved unnecessary, however, only when it is performed after the expulsion of the last stone; even then the operation will not have been absolutely unfruitful, for the demonstration of the true condition will not be without value. In my experience there have been but one or two cases in which the last colic occurred during the expulsion of the last stone. If the diagnosis is clearly established by the discovery of a stone in the stools, or if the facetting of that stone indicates that others remain, exploration is advisable, even with the chance that the last stone has escaped. The

discovery of a non-facetted stone does not disprove the existence of other non-facetted stones. I have found many stones, especially of the finely nodular variety, without a single facette. The slight chance of operating after the escape of the last stone must always be taken. The exploration may be proved unnecessary, too, by the discovery of hopeless disease,—cancer of the pancreas, stomach, colon, or liver. The demonstration of hopelessness, however, is not without value—as, indeed, the demonstration of an empty gall-bladder is not without value. To discover that the last stone has escaped, to demonstrate in malignant disease that nothing remains to be done, these truths surely justify the slight risks of exploration. For one such example of needless or of hopeless intervention, there will be many permanent cures.

(3) Hernia in the scar may result.

In simple explorations, in the separation of adhesions, in operations which open neither the biliary nor the alimentary tract nor a septic focus, the abdominal wound can be closed immediately, and hernia will rarely result. Wounds which must be drained for a few weeks are not infrequently followed by hernia, which results partly from retraction of the oblique and transversalis muscles, partly from division of motor nerves. Hernias high up in the abdomen, however, rarely cause serious disability. Gravity diminishes the intra-abdominal pressure upon the scar, and it is only in straining that the bulging appears. The liability to hernia, is, however, a disadvantage.

(4) There is the possibility that gall-stones may recur.

This subject is one of great interest. The conditions which led to the formation of gall-stones in the first place would naturally lead to their reformation. If they were in the first instance caused by infections of bile, it would be necessary for their re-formation that there be a renewed infection. Such a recurrence of infection would, however,

seem on the chances unlikely to take place. That gall-stones are originally thus dependent, seems to have been shown by the discovery of micro-organisms in their centre—a fact which has already been noted by others, and which in my cases has been demonstrated by Dr. Mark W. Richardson. If the germ origin of gall-stones is true, the advisability of cholecystotomy and drainage is unquestionable; for thus would be insured not only the expulsion of the gall-stones, but of the prime cause itself. Indeed, drainage at the time of primary infection would probably have prevented in the first instance the formation of gall-stones. I have never seen or had reason to suspect the recurrence of gall-stones after cholecystotomy and drainage. This argument against intervention seems, therefore, to have little weight.

(5) There is the possibility of spontaneous cure.

That gall-stones may escape completely and permanently through natural processes, cannot be doubted. That this fact should have great weight as an argument against intervention when gall-stones begin to offend, is, in my judgment, contraverted by experience and by common sense.

The question of spontaneous cure is a purely mechanical one. Certain gall-stones permit a safe spontaneous cure, and only certain ones,—namely, those small enough and smooth enough to pass through the cystic and common ducts, and through the duodenal papilla, into the duodenum. All the gall-stones must be no larger than this certain size to be safely evacuated. In some instances, however, even when the stones are all of about the same size, and small enough to be expelled, only a few will escape with each attack of biliary colic. In a case in which I saw a dozen or more gall-stones, about as large as a medium-sized pea, pass during one attack, I subsequently opened the gall-bladder and found large numbers of similar stones. In some cases I have found a great number of gall-stones of

the same shape and size, with some very large ones. There may be one or two as large as an olive or a filbert, many the size of a pea, and many extremely small. The expulsion of the smaller stones may take place without symptoms; of the medium-sized, with repeated attacks of biliary colic; but expulsion of the larger ones can take place only by direct ulceration through the gall-bladder walls. The patient may live for years, and undergo numerous attacks of colic, without impaction of the larger stones. No one can predict, however, even when numerous small stones have been demonstrated in the stools, when the impaction of a large stone will cause a permanent jaundice. Many stones may exist in the gall-bladder without offending. While they are in the gall-bladder, however, we must always be prepared for infections of the gall-bladder through the irritation which they cause. The emergencies of acute gall-bladder infections are by no means uncommon. They occur in a large proportion of cases without previous evidence of their presence. The very first symptom may be a fulminating infection of the gall-bladder, resulting in gangrene, —it may be a sudden perforation, with extensive and fatal flooding of the whole abdomen. Though we cannot prevent the occurrence of such disasters when gall-stones are unsuspected, we can anticipate them and provide against them when they are suspected, and that, too, at a very small risk.

Spontaneous cure through the dangerous process of adhesive peritonitis and evacuation into the intestine is possible. I have already mentioned the escape of a gall-stone into the intestine, with complete intestinal obstruction, in a man who had never had a moment's pain. The stone may in rare instances escape through the abdominal wall. For one recovery through so clumsy and dangerous a method of spontaneous mechanical cure, however, there are many deaths. In relying upon it, the physician relies upon weak-

ness, when, by the art of surgery, he might rely upon strength.

Once gall-stones begin to offend, then, the only possibility of spontaneous cure lies in the successful expulsion of the stones through the biliary passages, or in the successful ulceration into the alimentary canal,—or, in very rare instances, in the successful ulceration through the abdominal wall.

The foregoing statement of facts concerning the spontaneous cure of offending gall-stones includes the only argument of importance against surgical intervention. But we have, under the most favorable circumstances, painful, perhaps often repeated and prolonged colics, with a strong probability that they will continue indefinitely, with no certainty of permanent cure,—with every probability that with increasing age of the patient and increasing size of the stone, the difficulties and dangers of passage will also increase.

We have on one hand the painful, dangerous, and uncertain processes of ulceration,—processes which may require years for their completion, and which invite the disasters of localized infection, biliary extravasation, general peritonitis, faulty adhesions, liver and pancreatic infections, and new growths,—the clumsy, dangerous, and ineffectual methods of nature. We have on the other hand the safe, rapid, intelligent, and scientific work of a few minutes, with absolute demonstration, not only that the gall-stones themselves have been removed, but that complicating conditions have, if possible, been relieved.

(6) There is also the possibility that, after offending enough to prove the diagnosis, gall-stones may give no further trouble.

The possibility that gall-stones which have once offended may never again offend, cannot be denied. The fact that in a given case it is impossible to tell what the future will

be makes this argument of little weight. If, in any case, there are distinct contra-indications to operation, the possibility that there will be no further trouble may be looked upon with a certain satisfaction; but, as an argument against intervention, this possibility should, receive but little consideration.

(7) The last and decisive attack of biliary colic may have been caused by the last remaining gall-stone, exploration showing that none remain.

This argument has already been discussed.

As time goes by, the difficulties and doubts which many of us felt in the beginning as to the best treatment of gall-stones become dissipated in the light of renewed experience. Aided by numerous explorations upon the living, especially in cases of doubt, we are becoming more and more accurate in diagnosticating the lesion. We have demonstrated, by thousands of operations upon the abdominal cavity, the safety of simple explorations. At the same time we have demonstrated in hundreds of cases the pathological appearances of gall-stone lesions in their earliest stages. We have seen the changes, after years of natural efforts at expulsion, in the thickened, contracted, and friable gall-bladder; in the gall-bladder buried in extensive adhesions of the right upper quadrant, contracted upon large stones, ulcerated, and filled with septic fluids. We have seen the perforations from the gall-bladder into the intestine, the peritoneal cavity, the abdominal wall; obstructions of the cystic, hepatic, and common ducts; strictures at the duodenal papilla; biliary fistulæ; the acute infections of the gall-bladder varying between simple distentions with mildly septic fluids and total gangrene of the gall-bladder wall. We have seen the gall-bladder distended with the chronic puriform fluids of long continued inflammation, or infected by the highly septic fluids of an acute cholecystitis. We have observed, only too often, malignant new growths of the interior of the

gall-bladder itself; cancer of the liver secondary to cancer of the gall-bladder; acute and chronic infections of the pancreas. In a word, we have seen a great variety of lesions, owing directly or indirectly to the presence of gall-stones, lesions which in the early stages would have been safely prevented by surgical treatment.

The spontaneous cure, whether or not it is aided by medicinal treatment, is clumsy, dangerous, ineffectual, and contrary to common sense. Once gall-stones begin to offend, there is no way of telling how the process will end, except that, on the chances, it will end either in permanent disability or in death. Treated mechanically, as they are by surgical art, gall-stones are quickly, safely, and permanently removed, at the least possible expense in suffering and in danger.

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## DISCUSSION.

Dr. A. H. TUTTLE, of Cambridge: There are only a few points to be considered in regard to the advisability of operating in every case where gall stones are found. When gall stones are present and it is plain that they are going to undermine the health of the patient, I agree with Dr. Richardson that operation is necessary. But experience shows us that gall stones can be present for years without injuring in any way the health of the patient, and in some cases their presence is not even suspected. Again, operations on the gall bladder do not always turn out so satisfactorily as we would wish for. The operation may appear to be perfectly simple. I remember one such case in my own experience where it was not a success. In other words, the operation is one which we cannot say will always turn out all right. How can we tell just what conditions we are going to find when we open the abdomen? On the 4th of July, more than 24 years ago, I had my first experience with gall stones. The attack was a simple one, and the symptoms promptly subsided. In 48 hours a second

attack occurred. It was relieved in the same way. And for ten years no other symptoms of gall stones occurred. Then a slight manifestation, and until now there has been no recurrence.

A few years ago I was called to perform an autopsy on the wife of a physician. This physician and his wife had been married a great many years, and during that time there was never any symptoms that would lead him to think that his wife had gall stones. At the autopsy a very large gall stone was found, which must have been present many years, without giving rise to any trouble whatever so far as we could find out.

A gall stone in the bladder does not necessarily produce all the symptoms pointed out by Dr. Richardson. Personally, I would not want to say that every time I discovered a gall stone I would advise operation. I have seen too many cases get along perfectly well without an operation. It is not an operation like that for appendicitis. The day has not come when we can go in and remove the gall bladder. When we can do so then I think it will be advisable to operate for all cases of gall stones. In appendicitis we remove the appendix, and that is the end of the matter. After opening the gall bladder and removing all the stones contained therein, what guarantee have we that more stones will not form?

ARTICLE XLVI.

# THE PUPIL IN GENERAL DISEASE.

By EDWIN E. JACK, M.D.,
OF BOSTON.

READ JUNE 11, 1901.

# THE PUPIL IN GENERAL DISEASE.

For the recognition and correct interpretation of pupillary symptoms it is necessary that there be a clear understanding of the normal actions of the pupil, the mechanism of those actions, the proper method of testing them and the variations within the normal. It must be borne in mind, too, that abnormal pupils are often caused by pathological lesions local to the eye.

The normal movements of the pupil are reflex and associated. The reflex movement is contraction under the influence of light falling on the retina and dilatation on the removal of the light. The associated action is the contraction in the accommodative effort, the focusing from a distant to a near object, and in the converging movement of the eyes in fixing that object.

The nerves governing the iris are the third and the sympathetic. The stimulus of light falling upon the retina is transmitted through the optic nerves and tracts branching by some path to the pupil-contracting nucleus of the third nerve in the floor of the third ventricle and from here out to the iris. The associated contraction is brought about by the coincident action of this centre and those for convergence and accommodation which are near it. The active pupil dilating centre is the so called ciliospinal centre in the sympathetic system in the lower cervical cord. It is considered that the normal moderate dilation of the pupils is dependent on the constant psychical and sensitive stimuli

transmitted through this nerve to the iris,—the sympathetic reflex.

In testing for the light reflex it is a common error that the patient is looking at the source of light or at some near object, thus bringing into the test the contraction associated with accommodation. The eyes, tested one at a time, the other being covered, should look at a distance of twenty feet or more into darkness or at some object. The source of light should be in front and slightly to one side, a condensing lens being used in doubtful cases; direct daylight may be used. If perception of light only be present the reflex will take place. The pupil of the second eye will also react to the light thrown upon its fellow because of the connections between the two sides. A blind eye will not react to the light thrown on its own retina, nor will the second eye, but both will react perfectly if light be thrown on the other, sound eye. There is a rare exception to the rule that the pupils of blind eyes will not react, and that is when a lesion is high up in the optic tracts beyond the reflex arcs, as *e. g.*, in uræmia. There are, too, frequent cases in which seeing eyes give no reaction, mention of which will be made later.*

There is no absolute normal standard for the size of the pupils. They vary of course in different lights; they vary in different individuals, both in size and activity, and according to age. Babies have small pupils, young and middle aged larger and active ones, old people small and sluggish ones on account of the more unyielding nature of the iris tissue. They are larger in myopia. The pupils are contracted in sleep because then all psychical and sensitive stimuli are in abeyance.

Mention may best be made here of the dilatation of the pupil in psychical emotion, fright, anger, in painful sensory

* The reaction in accommodation and convergence is readily demonstrated by directing the patient's gaze from a distant to a near object (the finger *e. g.*) in the same line.

impressions, in fatigue, in dyspnœa, deep inspiration and with labor pains, through the agency of the sympathetic. The dilatation in anæsthesia is a warning. Patients with tobacco amblyopia have small pupils from the toxic action of the nicotine on the contracting centre, and watch makers and other people whose daily work compels constant close looking may develop miosis:—all these taking place in both eyes. One other reflex, that from irritation of the eyeball itself, should be spoken of. A foreign body on the cornea, *e. g.*, will cause the pupil to contract.

Irides bound down by adhesions to the anterior capsule of the lens may cause a pupil to be small and irresponsive to light, or the dilatation and contraction may take place only in a part or parts of the circumference. Chronic non-inflammatory glaucoma causes dilated and irresponsive or sluggish pupils, without other visible signs of disease,—a paralytic mydriasis from pressure on the fibres of the nerve. The same is seen in orbital disease, and after contusions of the eye, lasting long after the injury. General disease may cause blindness, and that in turn give loss of pupil reflex, as for example in the optic atrophy of tabes, or after gastro-intestinal hemorrhage.

Mydriatic and miotic drugs, as belladonna and pilocarpine, may act through the general system as well as locally. I have seen a paralysis of the pupil and accommodation follow the taking of frequent doses of Hilton's specific No. 3, so largely advertised, and a moderate degree of paralysis after a large amount of A. S. & B. pills, the effects of course being in both eyes. The importance of recognizing the local action of these drugs is often seen; patients using others' eyedrops in one or both eyes for some trivial trouble, not apparent at the time of the examination, and in some cases denying their use. The applications in others are accidentally made by the fingers or towel, or dropper which has been used for atropine. Ptomaines (fish, meat, etc.)

may cause dilated pupils with paralysis of the accommodation.

Coming now to the effect of general disease on the pupil, it will be inferred from the nature of the nervous supply of the iris that dilatation may be paralytic, third nerve, or irritative, sympathetic; and that contraction may be paralytic, sympathetic or irritative, third nerve. With this in mind the variations of the pupil in disease can be more readily understood and a detailed account of the many pathological conditions which cause pupil changes becomes unnecessary. Speaking in a general way, paralytic mydriasis is seen in progressive paralysis where at first there was miosis, and in various diseased processes at the base of the brain affecting the third nucleus or nerve. Irritation mydriasis occurs in spinal meningitis of the cervical portion of the cord, in the spinal irritation of anæmic or chlorotic patients, after severe illness, as a premonitory sign of tabes, from intestinal worms, psychical excitement, *e.g.* acute mania, melancholia, in general paralysis, often then in one eye with miosis in the other. Unilateral mydriasis occurring at short intervals, first in one eye and then in the other, is, according to Von Graefe, a premonitory sign of mental derangement. It is said on high authority that unequal pupils are always pathological, but I have seen cases in which an inequality has lasted for years from childhood up without other symptoms developing. They are said by some to occur in hysteria. Irritation miosis occurs in the early stages of inflammatory affections of the brain and meninges, later there is dilatation in the stage of depression. Paralytic miosis occurs in spinal lesions above the dorsal vertebræ, *e. g.* tabes, in injuries, inflammation, etc. In acute mania, if miosis comes on, general paralysis may be prognosticated.

The lesions in these cases of course affect one or both sides, and in many instances involve other twigs of the third and other nerve branches.

There are two pupils which demand more than passing notice, the Argyll-Robertson and Wernicke's. In the first reacting in accommodation and not to light, the reflex arc is broken somewhere probably between the place where the optic fibres leave the tract and the third nucleus. This is a very frequent and early symptom in tabes; with it the pupils are usually small, even pin-hole (paralytic miosis, spinal miosis), but may be normal or even dilated, also of unequal size and oval as is common in miosis. The sympathetic reflex is lost later, and later still the reaction in convergence. The same phenomenon is present in general paralysis, but less frequently and usually later. In this disease pupil symptoms are frequent and early, beginning in most cases as an inequality; the same being present in other forms of insanity. It is an important question whether the Argyll-Robertson pupil is ever present without meaning the presence or approach of serious nervous disease, and the best authorities say that it never is. It is thus of very decided value.

Wernicke's symptom, the hemianopic pupil, is of important localizing value when it can be demonstrated. Light thrown on the blind half of an hemianopic eye will still cause reflex if the lesion be above the reflex arc in the brain, leaving the arc intact, but if it be in the tract below the point where the optic fibers branch to form this arc there will be no reflex. As an exception, however, the pupil may react when the lesion is in the tract, the special fibres for the reflex being perhaps more resistant than the visual fibres. It is difficult in testing for this reaction to limit the light to the blind half of the retina, and the theoretical value of the test is not always realized. It is usually a more sluggish pupil that is found and not a total lack of reflex. Lesions causing this pupil are not necessarily limited to the tract, neighboring processes may cause it by extension or

pressure on the tract. Lesions at the chiasma have also been observed to cause this hemianopic reaction.

A peculiar form of pupil movement known as the paradoxical pupil symptom is the slight oscillation of the pupil when light is thrown into the eye, so-called hippus, which rapidly goes on to dilatation instead of contraction. It is considered an early sign of coming paralysis, but is also reported in hysteria and disseminated sclerosis. Investigation into the state of the pupil in the algid period of cholera has shown that if the light reflex is present prognosis is good, if absent it is bad; the size does not aid. In epilepsy conditions vary, but there is usually contraction at first and dilatation later, the light reflex being lost, this point being important in distinguishing the attack from hysteria. (I am positive, however, that I have seen dilated and irresponsive pupils in hysteria.) Bilateral and unilateral mydriasis have been noted in diabetes.

The question naturally arises, of how much value are pupil symptoms. In diagnosing the situation or nature of an intracranical lesion they are, if alone, of but little significance except that the lesion must affect the nucleus itself or be peripheral. If, however, with this isolated symptom there are those from the involvement of other branches or nerves the importance may become considerable. The early period at which some pupil changes come on in organic nervous disease, often being the first symptom seen and, it may be, of characteristic form, gives them much value. Mention may be made of the dilated and irresponsive pupils of brain compression after injury which call for interference. If we could take as absolute the difference in the pupils in epilepsy and hysteria, apoplexy and alcoholism, etc., we would have valuable data, but authorities do not wholly agree, and I have no personal experience to give. About the great importance of the Argyll-Robertson and

Wernicke's pupil of course there can be no doubt, and the value of pupil symptoms as an addition to others is often experienced.

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## DISCUSSION.

Dr. E. L. PARKS, of Boston: I am very much interested in what Dr. Jack has said in his paper, but have been looking for one thing which I expected him to mention, viz., the diagnosis of compression of the brain and concussion of the brain. I have always thought that it was a constant symptom in concussion of the brain that the pupil is contracted, and that in compression of the brain the pupil is dilated. I have felt that this was as reliable as any one symptom I ever found. I would be very much obliged to Dr. Jack if he would say whether he has observed this or not.

Dr. JACK: I have no experience in these cases, but I believe that there is no hard and fast rule in regard to this nowadays; it has been given up. It is true that concussion of the brain usually does carry with it a contracted pupil, and compression of the brain a dilated pupil, but I believe no absolutely definite rules can be laid down.

ARTICLES XLVII.–LII.

# THE DISEASES OF NUTRITION OF INFANTS.

## GENERAL CONSIDERATIONS.

By THOMAS M. ROTCH, M.D.
OF BOSTON.

## INFANTILE ATROPHY.

By JOHN L. MORSE, M.D.
OF BOSTON.

## INFANTILE SCURVY.

By EDWARD L. PEIRSON, M.D.
OF SALEM.

## RHACHITIS.

By ARTHUR R. CRANDELL, M.D.
OF TAUNTON.

## ASSOCIATION OF ANÆMIA WITH CHRONIC ENLARGEMENT OF THE SPLEEN.

By ARTHUR H. WENTWORTH, M.D.
OF BOSTON.

## CRETINISM.

By CHARLES S. MILLET, M.D.
OF BROCKTON.

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ARTICLE XLVII.

# THE DISEASES OF NUTRITION IN INFANTS.

By THOMAS M. ROTCH, M.D.
OF BOSTON.

I HAVE been asked to make a few remarks introductory to the general subject of diseases of nutrition in infancy. There is a group of diseases which occur so essentially in that early period of development designated empyrically as infancy that it may be considered to be characteristic of that period. This group consists of four diseases, *infantile atrophy*, *infantile scorbutus*, *rhachitis*, and *osteomalacia*. Of these diseases, infantile atrophy and rhachitis are essentially morbid processes occurring in the first two years of life. Infantile scorbutus also while having its counterpart in the scorbutus of a later period of development both in its pathology and in its treatment, yet shows in its symptoms and course such a characteristic picture in the middle period of infancy that it may be considered at least as being a special form of scorbutus. While it is necessary to include osteomalacia in the diseases of nutrition of infancy, it, unlike the other three diseases, only occasionally occurs in children, and is more a disease of adult life. It, however, at times simulates so closely in its manifestations rhachitis that it should be included in the group, but it is not a disease characteristic of infancy as is the case with the others.

The etiology of this entire group is very obscure, but each member of the group has its own peculiar manifesta-

tions which separate the members from each other, classify them under one head, and present clearly a picture of a vice of nutrition.

This group of diseases is assuming a more and more important position among the morbid conditions belonging to the early years of life, as it is becoming evident that it is far more reaching in its effects, and influences to a much greater degree all the diseases of early life than in former years was recognized as possible. Thus the prognosis of all those diseases which have a definitely determined etiology is markedly graver where such diseases occur in an infant who is suffering from one of these general disturbances of nutrition, and in this way especially both rhachitis and infantile atrophy have come to play a great and important role in our study of infantile diseases in general and in their therapeutics. When we consider the high mortality of these diseases when uncomplicated, and the greatly increased mortality of all diseases which are complicated by them, and when we recognise that these diseases are closely associated with improper food and hygeine, it becomes very evident that the proper therapeutic management of this group of diseases is of very great importance and should demand especial study and care in the feeding in the early months and years of life. There is no doubt that if less attention was paid to the use of drugs in the various non-organic disturbances of the gastro-enteric tract in early infancy, and that if a more enlightened method of feeding were adopted by laymen and physicians, or rather by laymen through the more intelligent and advanced students of this subject, it would be possible to eradicate this entire group from its place among the various pathological conditions of early life, and that thousands of lives might easily be saved where they are now recklessly thrown away. This fact is explained by the strong position which theoretical ignorance holds in comparison with that practical and scientific know-

ledge which exists and is only waiting to be received and to be given a fair trial in order to prove its wisdom and success. The field for purely dietetic treatment is an immense one, and such treatment will in the future, while supplanting the older methods of treatment, and we might say the causes of these nutritive diseases, blot them out from our nomenclature and make their pathology a museum relic of the past.

It is therefore well worth the time and thought of this Medical Section of The Massachusetts Medical Society to discuss in detail the different members of this protean group of diseases, for a thorough knowledge as to the diagnosis and treatment of each one of them is of such vast importance to us physicians who, while our mission is to cure, still hold as the highest object and aim of our art the study and practice of prophylaxis.

The three important members of this group of diseases, infantile atrophy, infantile scorbutus, and rhachitis, will presently be spoken of The fourth and rare disease, osteomalacia can be described in a very few words.

Nothing definite is known about the etiology of osteomalacia and it is simply spoken of in connection with diseases of nutrition on account of its resemblance to rhachitis. There is, according to Ziegler, an absorption of lime salt beginning first at the medullary cavity and proceeding outward. The epiphyses are not notably affected by the continuance of the absorptive process. The cortical bone becomes spongy and decalcified, and in the severest cases there may remain little but marrow and periosteum. The opinion is generally held that in osteomalacia the layer of osteoid tissue results from decalcification, while in rhachitis a similar layer represents a new growth deficient in lime salts. The periosteum is likely to be thickened and vascular, and the medulla resembles that of infancy in its gross appearance. Spontaneous fractures and various distortions

may occur in osteomalacia, and the thorax is flattened laterally. The treatment of osteomalacia is the same as in rhachitis, and is essentially dietetic and hygienic. In regard to the diet the special age and development of the individual should be considered. Care should be taken not to force into use the functions of digestion and absorption, but to allow them to gradually develop until at the time indicated by nature they are ready normally to assume charge of the special elements of the food which it is their peculiar province to digest and to assimilate.

ARTICLE XLVIII.

# INFANTILE ATROPHY.

By JOHN LOVETT MORSE, M.D.
OF BOSTON.

The term, infantile atrophy, and its synonyms, athrepsia and marasmus, should be applied only to those cases of wasting which, in the light of our present knowledge, are primary and not secondary to other morbid processes. These cases of primary wasting form a small but perfectly definite clinical group, easily distinguishable from the more common cases of wasting secondary to other diseases and conditions.

*Definition.*—Infantile atrophy is a morbid condition of infancy in which there is extreme wasting of the soft tissues of the body without demonstrable organic lesions. It is the expression of continuous, insufficient nutrition due probably to defective absorption or assimilation rather than to defective digestion.

*Etiology.*—It occurs most frequently during the first six months of life, less frequently during the second six, and is rare after the first year. It is more common in cities than in the country, and among the poor than among the well-to-do. It almost never occurs in breast-fed infants. Prematurity, inherent weakness of constitution, lack of warmth, light, pure air and cleanliness are most certainly predisposing factors. Unsuitable food undoubtedly favors its development. In a certain number of cases it follows or is

engrafted on severe intestinal disturbances, whether functional or organic. None of these things, however, afford a satisfactory explanation as to the origin of the disease. They are all common, atrophy is rare. They all give rise to other conditions as well as to atrophy. Therefore some other factor, presumably common to all cases and more important than these, must be present. This factor has not yet been discovered. The wasting of the tissues, which is the chief symptom of the disease, must be due, in the absence of demonstrable lesions, to an insufficient supply of nutriment. As in typical cases the processes of digestion are apparently carried on normally, the source of this insufficient supply of nutriment must be some disturbance of the functions of absorption or assimilation. The nature of this disturbance of function and the causes, except as imperfectly detailed above, are at present unknown, the examination of the urine and feces having thus far failed to furnish any information of importance as to the character of the disturbances of either absorption or metabolism.

*Pathological Anatomy.*—The pathological findings afford little or no information as to the nature of the disease. All the tissues are dry and the fat is almost entirely wanting in the skin and about the internal organs. There is extreme atrophy of all the muscles. The heart is, as a rule, normal but may be fatty. The liver is not infrequently enlarged and fatty. The kidneys are sometimes fatty and often contain uric acid infarctions. These fatty changes in the various organs are undoubtedly merely secondary manifestations of the impaired nutrition. There is marked atrophy of the mucous and submucous coats of the intestines. Microscopical examination, however, fails to show any characteristic lesions. The superficial lymph nodes are, as a rule, enlarged. The retroperitoneal and mesenteric nodes may be, but if so, show no abnormal changes microscopically. The lungs often show areas of atalectasis or the

lesions of complicating bronchitis or broncho-pneumonia, while the intestines may show those of a complicating catarrhal or follicular inflammation.

*Symptoms and Course.*—The one prominent symptom of infantile atrophy is failure to gain or loss of weight. This is the earliest and for a time may be the only symptom. It is constant and progressive. In the typical case there are no symptoms of disturbed digestion. The appetite is, as a rule, diminished but may be voracious. Thirst is increased. Vomiting is unusual. The stools are normal or constipated. The child is usually quiet and apathetic, but may be peevish and irritable. The temperature is normal or subnormal. Sooner or later complicating functional disturbances of digestion or inflammatory conditions of the gastro-enteric tract arise and add vomiting, diarrhœa, colic and fever to the list of symptoms. These may be acute or chronic, temporary or lasting.

The picture of an advanced case of atrophy is very characteristic. The fontanelle is small and shrunken. The bones of the skull overlap. The face is pinched, the chin pointed, the bones prominent and the skin wrinkled, especially about the mouth and forehead. A cry is distressing; a smile, dreadful. The eyes are wide open and staring or half closed and dull. The cry is feeble and the movements slow and infrequent. The tongue is thickly coated or dry and red, while the mucous membrane of the mouth may show herpetic lesions or those of thrush. The skin, everywhere wrinkled, yellowish, dry and desquamating, hangs in folds from the bony prominences, as if draped over the skeleton. Pustules on the scalp and body, and intertrigo about the genitalia and buttocks are common, while there is often an excessive growth of lanugo on the neck and back. Ecchymoses occur not infrequently. The abdomen is sunken and lax. The extremities are cold, the hands look like claws, and the muscles like strings. Rigidity of the extremities is not uncom-

mon, presumably as the result of the cerebral anæmia. Œdema sometimes occurs. Chronic otitis media is not infrequent.

Emaciation continues, the strength steadily fails, and finally the child becomes too feeble to suck. The action of the heart becomes weak and the circulation poor. The pulse is feeble and rapid, although sometimes slow. The extremities become cold and cyanotic and pulmonary atalectasis develops. The respiration is weak and superficial and the temperature subnormal. Death finally occurs after gradual, progressive failure. In some instances sinus thrombosis results from the feeble circulation and death may be preceded by spastic phenomena or convulsions. Functional and inflammatory disorders of the gastro-enteric tract are frequent and serious complications. Bronchitis and broncho-pneumonia are not unusual complications while an acute, general, bacterial infection often brings the disease to a sudden close.

The urine is usually diminished in amount and contains an excess of both urea and uric acid. It not infrequently contains albumin and sometimes sugar. The blood shows the conditions ordinarily found in secondary anæmia. In some cases there is also a moderate increase in the number of white corpuscles.

*Diagnosis.*—The diagnosis is from starvation, wasting secondary to functional or organic disease of the stomach or intestine, congenital syphilis and disseminated tuberculosis.

The diagnosis from starvation due to congenital deformities is self-evident. That from starvation due to insufficient but suitable food is also plain. The rapid gain when sufficient food is given will settle any doubt. That from starvation due to food of improper character can usually be readily made by a careful consideration of the food in question and by the rapid improvement when a suitable food is given.

The diagnosis from gastro-intestinal diseases associated with wasting must often be a difficult one, as infantile atrophy is often complicated by them. In them, however, the wasting is not the one prominent symptom as it is in infantile atrophy. It is, moreover, not the earliest but a late symptom, always following other symptoms of gastric or intestinal disturbance. Vomiting and diarrhœa are more common. The stools show evidences of indigestion and inflammation not present in those of infantile atrophy. The abdomen is, as a rule, distended. The temperature is usually elevated. There are fretfulness and sleeplessness instead of apathy. The course is not so progressively and uninterruptedly downward. Infantile atrophy in the vast majority of cases occurs in the first six months and rarely after the first year; these others occur at any age.

The history of syphilis in the parents and the presence of other signs of syphilis, active or inactive, make the diagnosis from syphilis with emaciation easy.

The diagnosis from disseminated tuberculosis, however, is frequently difficult and at times almost impossible, for in the tuberculosis of infancy the local symptoms are, as a rule, subordinate to the general. A family history of tuberculosis or of continued exposure is of certain but not of great importance. So also is a doubtful milk supply. The temperature is likely to be elevated in tuberculosis, but is not always. On the other hand, some cases of infantile atrophy, especially if complicated, have fever. Diarrhœa cannot be considered as pointing especially to tuberculosis of the intestines as it may as well be due to some complicating condition. Enlargement of the spleen is in favor of tuberculosis. Enlargement of the liver may occur in either. Râles are almost always present in the lungs of cases of atrophy as the result of congestion or œdema or of a complicating bronchitis. Solidification of the lung is the most important point in the differential diagnosis except in the

rare instances in which tubercle bacilli can be demonstrated in the sputum or feces. Even solidification, however, may be due to a complicating broncho-pneumonia.

*Prognosis.*—The prognosis is always grave. Most of the cases result fatally. If recovery takes place it is always slow and is usually interrupted by frequent relapses. When recovery takes place, however, it is complete, and the future development is not interfered with. Complicating functional or inflammatory disturbances of the gastro-enteric tract render the prognosis still more grave, as do diseases of the respiratory tract. Other diseases occurring in infants with atrophy carry a very bad prognosis. Secondary general bacterial infections always result fatally.

*Treatment.*—There is no known drug which has any specific action in infantile atrophy. As the condition is presumably due to some vice of absorption or assimilation the treatment must necessarily consist largely of regulation of the diet with the object of providing some food which can be easily absorbed and utilized by the individual infant. The same food will not fit every case. The best food and the one which is always to be tried first, if circumstances permit, is human breast milk. The best substitute for it is some modification of fresh cows' milk, prepared either at the laboratory or at home. Although there are no data, based on scientific experiments, to suggest in what way cows' milk should be best modified for these cases, clinical experience has shown that, as a rule, they do best on milks which contain a low percentage of fat, a moderate percentage of sugar and a moderate or somewhat high percentage of proteids. For some unknown reason they seem unable to utilize any but small amounts of fat and continue to lose if larger quantities are given. Hence the treatment by larger doses of cod-liver oil is not only useless but harmful. They do seem able, however, to make use of considerable amounts of proteids. The proportions which are best suited for the

early treatment of the majority of these cases lie within the following limits:—

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|---|---|
| Fat | 0.50 to 1.00 |
| Sugar | 4.00 " 5.00 |
| Proteids | 1.00 " 2.00 |

Whey mixtures, furnishing, as they do, a considerable proportion of the proteids in the form of the more easily digestible and absorbable lactalbumen, are especially useful in giving these high percentages of proteids. Unfortunately their preparation outside of the laboratory is decidedly difficult. A portion of the proteids may also be added to the milk mixtures in the form of egg-albumen which is more easily absorbable than the milk proteids. Peptonizing the milk-mixtures is also often useful. While the preparations given above are those best suited to the average case, many do not do well on them. In such cases various modifications must be tried until one is found which suits the individual infant. Unfortunately it is not infrequently impossible to find such a modification, and the infant continues to lose and finally dies. In cases which improve it is usually possible to gradually increase the amount of fat in the food and finally to work up to the proportions which are suitable for a normal child of the same age. In beginning treatment it is best to give small amounts of food at short intervals. It is often advisable to give proteids apart from milk. They are best given in the form of egg-albumen or beef juice. Somatose, in doses of five or ten grains, mixed with water, is also useful.

Due attention must be paid to cleanliness and to the maintenance of an unusual supply of fresh air and sunlight. It is especially important to keep up the body temperature as the vitality of these infants is very low. They must not be handled more than is absolutely necessary and all excitement must be avoided. No point in the hygiene and nursing of these cases is so small as not to be of importance.

Stimulation, preferably in the form of brandy or whiskey, is often necessary.

Complications must be treated as they arise.

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## DISCUSSION.

Dr. C. P. PUTNAM, of Boston: Dr. Morse has given us a brilliant and interesting picture of this strange condition, which is always appalling and often fatal. It has always been recognized that among infants that die a certain number simply waste away. The condition has been called marasmus. Certain symptoms, e. g., thrush, have been considered as essential parts or even causes of this wasting. But about a quarter of a century ago Parrot first collected together all the symptoms so graphically described to-day by Dr. Morse and many others beside, which he found in wasting infants, and to this picture he gave the name Athrepsia. This picture was so striking that since then no writer or any general treatise has failed to say something about Infantile Atrophy or Athrepsia. Parrot described in detail these many additional symptoms and pathological changes not as accidental, but as pathognomonic. But writers since then have gradually lopped off most of them as being merely complications, and like Dr. Morse have come to set aside cases of atrophy resulting from tuberculosis, starvation (from improper food), diseases of the stomach and intestines, syphilis, premature birth, etc.; in other words, all secondary cases of atrophy, in order to come at the primary or essential atrophy which is, as we have heard to-day, devoid of any demonstrable organic lesion and therefore totally inexplicable.

But this lopping process leaves so few primary cases that it seems to me the question is whether there is any such thing as a primary idiopathic atrophy. Certainly there is much to be said against the proposition.

In the first place, it occurs very seldom with infants fed under what we regard as normal conditions. That it sometimes starts with children on the breast does not seem to me

conclusive, for we well know that the human milk is not by any means always suitable to the child, sometimes because there is not enough milk, sometimes because the quality is not good.

In the second place, has it been our experience that a child starting well and brought up under our eye has become atrophic without our being able to say what the cause was? Certainly this has not been my experience.

Whether with children on the breast or with those on the bottle the foundations of this condition have always been laid while the baby was out of sight and out of mind of a physician accustomed to recognize the early beginnings.

It is not conclusive to say that autopsies have shown the absence of gastric and intestinal diseases that would fairly account for such a serious train of symptoms. It seems to be sufficient answer to this that very slight disturbances of digestion may produce in the delicate organism of babies such loss of function that a vicious circle is established from which there is in some instances no escape.

The very mildness of the symptoms of a digestive disturbance may lead one astray. A severe gastric or intestinal attack leads to vomiting or diarrhœa, and then we are not surprised to see the patient lose weight and vital force, but a slight indigestion may lead to nothing more than a slight autointoxication which leads to a lowered digestive and absorbing power, and this again leads to further autointoxication. So there gets to be a race between the absorption and the intoxication. Successful feeding, especially with the human breast, and such other aid as can be given, often wins the race, but often the feeding is unsuccessful and the race is lost.

For treatment, a good human breast, as has been said, is the best remedy, but if that cannot be found one must do what one can with bottle food.

That Dr. Morse is right in saying that a low percentage of fat and a relatively high percentage of proteids is desirable, I can neither affirm nor deny, but can only say that I have never thought so, but have always avoided a relatively high percentage of cow's casein, believing this part of cow's milk to be the most frequent cause of the malady. That the use of whey, whereby the casein of the cow may

be avoided may lead to a different conclusion, I think quite possible. Certainly, the fact that human milk plainly offers the best chance does not favor the theoretical use of a relatively high proteid and low fat, because the human milk has just the opposite composition.

As to any treatment beyond proper feeding, I will only mention washing of the stomach and colon, which is often of great service, and would be, I think, of still greater service if we were able to wash further in.

Dr. ABRAHAM JACOBI, of New York City: I have listened with a great deal of interest to the paper by Dr. Morse, and the accurate description of the symptoms has left nothing to add. From that point of view the paper is a master-piece. There were very few things which it seems to me could be added. One was, that in some of these cases the urine contains bacteria. Secondly, I notice that hydrochloric acid is absent from most of the stomachs of these babies. But not only is hydrochloric acid absent; all the juices are absent when emaciation has attained a high degree. Thirdly, as to thrush. It is always a bad symptom in any disease, where debility reaches the point of muscular incompetency of the mouth and cheeks and of deglutition. When this symptom is observed, the case, no matter whether typhoid, or any other disease which renders the patient powerless and unconscious, may almost always be set down as fatal simply because there is not action enough to get rid of the invading microbes, etc.

Amongst the points which determine the prognosis I will mention one which should not be forgotten. There is often the necessity when these patients are in the hospital to go back home, no matter of what description, away from the hospital, and often if this is carried out the patient will get well. It has been the practice in the large hospitals of this country and Europe to send them home, and that is often a turning point for a better condition of affairs.

Gain in weight. We are always glad to tell the mother when the child is gaining in weight. We must bear in mind that an atrophic baby may increase in weight because it is gradually becoming so full of œdematous fat that it is no good to it. Weight does not always mean health. Rhach-

itic babies, for instance, with their flabby feet and rotundity, often are heavy but not healthy in proportion.

I do not find that many of these cases will bear the beef juices. Many cases get worse if given the beef juices. A small quantity will become peptonized. The peptones change into propeptones, and give rise to putrefaction with foul stools.

Now in regard to the question,—What is atrophy, and is it a disease of its own? I am rather of the opinion just pronounced by Dr. Putnam. I have never been able to convince myself that there is an infantile atrophy that is a disease of itself, but I believe that it is simply a symptom of many diseases. It is an extreme waste from some well-known cause. I think we cannot from the beginning discriminate atrophy from "athrepsia" or "marasmus." When, in addition, we see a small, puny, poorly developed child, we are apt to make a diagnosis of atrophy, when it is simply aplasia. That means a congenital deficiency of cell growth, a reduction in the *number* of cells, while in atrophy it is not the number of cells that is faulty, but the wasting of the full number.

What are the causes of wasting in a little baby? It may be that it is a lack of vitality. Such babies have what is known as a lack of "germinal" vitality. These babies are small and remain small when they grow up. The baby may be at the breast, and there is what is alleged to be constipation, and the child wastes away. What is the reason for this? The baby is simply starving. It is not getting a sufficient quantity of the food it needs.

Hypertrophy or spasm of the pylorus may be the mechanical cause of atrophy. This is a much more common cause of atrophy than we are apt to think. I have seen a small number of cases during the past years. In these cases the symptoms are those of starvation. They take food, up it comes, they are not nourished, and have atrophy.

Another cause is cleft palate, which prevents the child nursing. These children look exactly like the cases described here today.

There are cases where anæmia is the cause of atrophy. Wherever there is anæmia the tissues are not well fed, the secretions are deficient, there is no motive power in the

muscles of the stomach and intestines, and no juices,—thus whatever interferes with the health of the child will cause anæmia and atrophy.

Then there are other cases with a distinct syphilitic history. These remain puny, and lose weight. These cases will never get well with arsenic and iron. The only treatment by which they will get well is by antisyphilitic treatment. These cases are seen not only in early infancy but also at the age of six or seven.

There are other cases, those caused by a diseased condition of the gastro-intestinal tract. Sometimes there is a local thinness of the mucous membrane and muscle. At some parts of the intestine round cell infiltration will be demonstrated, and there will be thin places where the mucous membrane is very thin and transparent, and places where the epithelium has come off. There is also a condition where there is a hyperplastic condition of the lymphatics of the mesentery, and the mesenteric glands will become swollen (rarely in tuberculosis, more frequently from simple intestinal catarrh, or fully developed enteritis) just as in some children the glands in the neck swell just as soon as they get a nasal or naso-pharyngeal catarrh. In those cases where there is round cell proliferation, disintegration finally results, and leaves the membrane in a thinned out condition.

There are different views in regard to the cause of atrophy. Huebner suggested that atrophy is the result of toxines. Well, in these days everything is caused by toxines or bacteria. Czerny says that atrophy is the result of an acid intoxication, exhibited first by the presence of ammonium in the urine. Tfaunder tells us that this acid intoxication does not take place except when assimilation, respiration and metabolism in general are seriously crippled, and that in every disease of the liver more ammonium and less urea are eliminated.

In comparing all these different views concerning etiology it has often appeared to me, that possibly many cases of atrophy, the causes of which we are unable to discover, will some day find their explanation in a defective development and faulty secretion of the ductless glands, the thyroids, thymus and suprarenals.

Another tells us that the best food for children is proprietary foods, and if you cannot get proprietary foods, milk, unmixed, is the best. Altogether we are fed in our literature on too many theories, obtained neither in the nursery nor in the sickroom, but by inexperienced men at their desks or in their laboratories.

Now and then a case of atrophy may be developed after malaria or a great loss of blood. There are some cases where we cannot find the cause at all. I repeat that I side with Dr. Putnam when he says that he does not think infantile atrophy is a disease of its own.

Dr. MORSE: I think that most of the criticisms of my paper were answered in the paper itself. I think that the gentlemen who have discussed my paper must have misunderstood the very careful and guarded statements which I made. The question has been raised as to whether infantile atrophy is, or is not, a disease. I am inclined to think that it is not a disease. I feel sure, however, that as I stated in my paper, there is a clinical group of cases, which, in the light of our present knowledge, are primary. This clinical group is so characteristic that it is deserving of separate consideration. It was of this group that I was speaking.

As I have just said, in the light of our present knowledge, these cases are primary. Dr. Jacobi has well shown our ignorance regarding their cause, enough, I think, to justify the statement I made that the cause is unknown. Whatever the cause may be, I think we are justified in saying that the condition is, that for some reason or reasons the outgo continues to be greater than the income. If the outgo continues to be greater than the income the child finally gets into a state in which the loss cannot possibly be made up and death results. The disturbance is presumably in the metabolism, but its nature is at present unknown.

As regards the pathology of the gastro-intestinal tract, it seems to me that Huebner's experiments have covered the ground very thoroughly and to me, at least, they seem satisfactory.

As to the treatment with low fats and high proteids which Dr. Putnam has criticized, I said very carefully in

my paper that this method of feeding was not founded on scientific experiments, but on clinical experience. Clinical experience in my hands has shown it to be more satisfactory than any other.

Dr. Jacobi has referred to the fact that beef juice increases the "stinking stools." I certainly agree with him that it does increase the odor of the stools when they are, as he says, "stinking." In the class of cases of which I am speaking, however, the stools are normal and not stinking. If they were the cases would not properly belong to the class which we are considering. In this class beef juice in moderate doses does not make the stools stink, and is, I feel, a very useful adjunct.

ARTICLE XLIX.

# INFANTILE SCURVY.

By EDWARD L. PEIRSON, M.D.
OF SALEM.

In taking up infantile scurvy, or scorbutus, as one of the diseases of the nutrltion of infants, I desire to present the subject to you simply in the light of a general practitioner seeing the case in his daily work, leaving the more doubtful points of pathology and etiology to be cleared up by the gentlemen discussing the subject.

There is no doubt that infantile scurvy is a distinct disease of childhood. There is no doubt that it is a fairly common disease and, unlike rhachitis, it is common among the well-tò-do families, where hygienic surroundings are good.

Two cases will serve to illustrate this disease.

Case I. Female child 13 months old. Child had been for some weeks under care of a physician, who was treating her for rheumatism, but she was constantly growing worse. She lay on her back in the crib with thighs flexed on body and knees flexed at right angles. The arms were kept flexed and close to body. Any attempt to move arms or legs caused the child intense pain, and in fact the child screamed in terror when anyone approached her. Her mother said she had been unable to change her diapers for four days. She was anæmic, poorly nourished, and weighed only twelve pounds at thirteen months of age. The mother stated she had been going down for three or four months,

and lately had been having nose bleed. On examination the lower ends of the diaphysis of the long bones of legs and arms were enlarged, especially the lower end of the femur and tibia and both bones of the forearm. The swellings were symmetrical and very tender. Fever was absent. There was no rosary, and the fontanelles were as much closed as age of the child demanded.

There were only four teeth and the gums were much swollen. There was moreover kyphosis and tenderness of spine. Under proper treatment there was marked improvement in four days, followed by complete recovery. This child had been fed exclusively on condensed milk.

Case II. Male child, ten months old, well nourished and fed on milk and barley in seemingly proper proportions. About two weeks before I saw him he slipped out of his chair and fell on the floor. About two or three days after his mother noticed that he did not use one leg much, that it seemed to hurt him to move it. This condition gradually grew worse until they sent for me. On examination I found a tender swelling over the lower end of the diaphysis of the femur and made a diagnosis of scurvy. Under treatment the swelling and tenderness wholly disappeared in six days.

These two cases are fairly typical of this disease and will serve for illustrations.

The etiology of the disease is doubtful. The most that can be said is that it follows the prolonged use of some diet unsuitable to that individual child, and, in general, the less like the food is to nature's food for the child at that age the more likely is scurvy to result.

The disease is met with usually in children under two years of age and generally from six to fifteen months. Sex makes no difference, and most cases that I have seen have been in native-born Americans, and in artificially fed infants. Proprietory foods and absence of fresh milk seem predisposing causes, but I have seen cases in infants who were

taking plenty of fresh uncooked milk. All that can be said is that either there is a lack of necessary elements, or else these elements are present in a non-assimilable form.

As far as the pathology, that which concerns us from a general practitioner's view, it is simply a hemorrhage that we have to deal with. The hemorrhage is the earliest symptom in the vast majority of cases, is usually subperiosteal and in the long bones of the legs, later in the arms, very rarely in any other bones.

The earliest and most common symptom is swelling of the lower end of the diaphysis of the femur—and this is an important point in the diagnosis from rhachitis, or rheumatism. In scurvy we find an extremely tender swelling at the lower end of the diaphysis, but the swelling is neither hot nor red. In rheumatism the swelling is in the joint and is hot. In rhachitis the epiphysis is enlarged and is not tender. If one bears these points in mind he will see more cases of scurvy than he thought existed before.

The rest of the pathology of the disease consists of anæmia which is probably secondary and due to the hemorrhage and a stomatitis ulcerosa. The swelling of the gums in my experience is not usually an early symptom and will not appear at all if the case is early recognized and treated. Hemorrhage may occur from the bowels, nose or into the skin later in the disease, or into any of the internal organs.

The first symptom that we notice when called to see a child with scurvy is pain. The child is in terror of being touched. When perfectly quiet there is no pain, but any motion, especially of the legs causes great distress. The legs and thighs are held flexed and rigid. The pain is first in the legs, then back and arms. The pain is early, constant and increases in severity as the case goes on. Next the swellings appear, and later the gums become spongy and ulcerated. There is no fever as a rule and the tender swellings are not hot. Anybody ought to be able to make a

positive diagnosis without much difficulty; yet most cases that I have seen have been mistaken for something else, and it is for this reason that I desire to present the subject to-day. The most common error in my experience is to call the cases rheumatism, yet rheumatism I should say, was extremely rare under two years, especially in the first six to twelve months of life when these cases of scorbutus appear. In rheumatism the joint is affected, it is hot and fever exists. In scurvy the lower end of the diaphysis is involved it is not hot and no fever is present. The next most common error is to lay the disability to some fall or injury which occurred at about the time the symptoms developed. I have seen quite a number of such instances where I was willing to say that if they would try orange juice for one week and the baby did not get well, I would acknowledge that they were right and my diagnosis of scurvy was wrong.

As regards the distinction between rhachitis and scorbutus there ought not to be any doubt. In rhachitis the swelling is epiphyseal, is chronic; is painless and by the time it is as large as the swelling of scorbutus, there will be a well-marked rosary, and the characteristic head deformities. Stomatitis and hemorrhages do not occur in rhachitis. The two diseases are at times found in the same child.

Pott's disease is rare under two years and has no swelling of the extremities. Acute anterior poliomyelitis has an acute and febrile onset with paralysis of later development. Purpura has no swellings of the extremities or stomatitis. Syphilis is more chronic and the swellings are less tender and therefore no one ought to fail to distinguish scorbutus from any of these four diseases. Practically if you are called to a child lying with legs drawn up, dreading to be touched, with swelling over the lower end of the diaphysis of long bones, anæmic, with swollen gums, there is no other diagnosis you can make except scorbutus and treatment from six or seven days will usually prove you correct.

Treatment is as a rule simple. The babies are usually bottle-fed and proprietary foods, condensed milk or cooked milk are used. Modified uncooked milk with orange or lemon juice should be given, one-half to one lemon or orange being used daily, with fresh beef juice. These children in my experience are greedy for the juice of both orange and beef. I usually give iron, etc., for the debility and anæmia. In severe cases the child must be kept quiet on a frame, but I have never had to resort to this as yet.

I am aware that in what I have said I have considered the subject from the somewhat superficial point of view of a general practitioner, but my object is to make it clear what a very easy disease scrobutus is, as a rule, to recognize, and also how many cases we all have probably overlooked in the past, and called something else.

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## DISCUSSION.

Dr. W. P. NORTHRUP, of New York City: Dr. Peirson modestly said when he read his paper, that it was not complete. I should say it was a very complete paper, and a good epitome of the subject of infantile scurvy as understood to-day; therefore, I will attempt only to say something upon certain features of the subject.

If you are going to cure a baby you must first make a diagnosis, and it is especially necessary to make the diagnosis of scurvy. It is very interesting to note that people are seeing cases all over the country. Some say they have seen a dozen cases. Some have had twenty cases, and so on. In 1889 a modest paper was written on infantile scorbutus, and read at a meeting in Washington at which Dr. Rotch was Chairman, and Dr. Osler was present. Dr. Osler picked up the paper and said, "Well, how many cases have you?" I answered "Eleven." He said that he had searched American literature for cases in preparing his book, but had found none. To-day Dr. Rotch probably has 150, while others have 100, and so on.

As to the etiology of this scurvy, I was much pleased with Dr. Peirson's words, viz., it is the persistent feeding of a child with something that is not suitable. I have seen scurvy develop in a child fed partly on the breast. We have not found that any certain food is entirely suitable for every case, but each baby has its own individuality, and needs its individual kind of food. It is the persistent feeding of food that is unsuited to the individual that causes the disease. It seems to me that the proprietary foods are responsible for the largest percentage of the cases of scurvy.

Now as to the symptoms. When a woman comes to you and says that the baby has rheumatism of the legs, and you find that it has spongy gums, *that child has scurvy*. They will say that the child has great pain, and (the earliest sign), cries pitifully when they diaper it.

Again, as to prophylaxis. I should say milk, good milk, and as near the mother's milk as you can possibly get it. The mother's milk is the best if it will fit, if not, one that comes as near the mother's milk as possible. As for the wet-nurse, she may or may not fit. I have had my troubles with these nurses, and have now come to depend upon the laboratory, where I can get the milk as I wish it. I have satisfied myself that we get better results than we do with the wet nurses we have in New York.

Treatment: The use of orange juice. It is very convincing I think, to see how these children with legs that cannot move will go for orange juice. If you have seen it once, you will think that orange juice is almost a specific for scurvy.

Dr. R. G. Freeman, of New York City: There has been a suspicion that the cooking of food might be an important factor in the production of scurvy in infants. This idea is readily reached by analogy from cases of the same disease in adults, which formerly occurred most frequently in sailors during long sea voyages, when fresh food was not available.

Although a case of scorbutus in infancy was reported nearly thirty years ago, the first general recognition of this disease in infancy occurred quite recently and at a time when milk was sterilized by long-continued boiling, and

many of the reported cases occurred during the use of this food. More recently Pasteurized milk has come under the same ban, for a certain number of cases of scurvy have occurred during its use.

Strangely enough this disease has been found most often in infants who were carefully fed and under the best hygienic surroundings, so that etiological factors common in many diseases may be excluded in most cases of infantile scurvy.

Barlow wrote recently in Grancher's System of Medicine, but before the publication of the collective investigation of the American Pediatric Society, as follows: "Experience seems to show that prolonged sterilization, and sterilization at a high temperature diminish the antiscorbutic properties of milk. For this reason we ought to practise sterilization or Pasteurization at as low temperature and for as short a time as possible and compatible with the destruction of pathogenic organisms." Booker, on the other hand, is quoted as saying after the collective investigation "I do not believe that sterilization of milk produces scurvy."

There have been now enough cases reported to enable one to draw fairly safe conclusions as to the importance of cooked food as a factor in the production of scurvy.

A study of the cases collected by a committee of the American Pediatric Society in 1898 would lead one to consider other factors than the application of heat and food as more important. Of 356 cases collected by them in which the food used when the disorder appeared is mentioned, only 16½% received sterilized milk, while 60% received proprietary foods, and only 4½% occurred during the use of Pasteurized milk alone (which was at that time much used), this being only slightly more than the nearly 3% of cases reported as occurring during feeding of breast milk alone and 3.4% occurring in babies fed on breast milk with other food.

Further light may be obtained by studying the change of food with which the treatment of scorbutus was inaugurated. Although in many of the cases raw food was given, we find that in four cases a change from sterilized milk to Pasteurized milk was made, in one from Pasteurized to sterilized, and in one from raw to sterilized.

If these statistics of the American Pediatric Society are to be accepted as reliable, and I believe they are as reliable

as any statistics can be, we must conclude that babies fed on Pasteurized milk have but little more tendeney to scurvy than breast fed babies; that children fed on sterilized milk have considerable more tendency to the development of scurvy than those fed on Pasteurized milk, while in the proprietary foods there exists an element which has the most powerful tendency to produce this disease, the nature of this element being as yet unknown. The only rational conclusion as it seems to me, is that scurvy is produced by the prolonged employment of an unsuitable food.

The food may be unsuitable either in quality or quantity. Some cases have seemed to be produced by the too prolonged use of too dilute modifications of milk, although experimental starvation in the lower animals has failed to produce scurvy.

That the prolonged application of a very high temperature to the food may have been a cause in some cases of rendering it unsuitable, cannot be denied, but it is evident that other more important causes of scurvy exist.

There exists no evidence that the Pasteurization of milk at the low temperature now known to be effective is an element in the production of scurvy.

ARTICLE L.

# RHACHITIS.

By ARTHUR R. CRANDELL, M.D.
OF TAUNTON.

Rhachitis is a chronic disease of nutrition.

It is essentially a disease of infancy, the symptoms usually appearing between the ages of six months and two years.

No race is free from its attack, but it is much more common among nationalities transported from warm to more temperate climates, as exemplified by its frequent occurrence in Negroes and Italians in this country.

Infants reared in the city are more prone to the disease than are the country-bred.

*Etiology.*—Various theories have been advanced to account for the origin of rhachitis, but they have been discarded one by one until it is now believed that this disease results from chronic malnutrition, often reenforced by poor hygiene and unsanitary environment. In support of this view is adduced the fact that breast-fed infants almost never develop rhachitis, unless lactation be unduly prolonged, or the breast milk be poor in quality. Artificially fed infants, on the other hand, frequently show evidences of rhachitis, especially when fed improperly. The diet in these cases is usually largely deficient in fats and proteids, with an excess of carbo-hydrates. This accounts for the common occurrence of rhachitis in children fed on proprietary foods, most of which contain low percentages of fat and proteids,

with carbo-hydrates in improper proportion, and often in improper form, as starch and cane sugar.

The influence of poor hygiene and unsanitary surroundings is shown by the frequency of rhachitis in city infants, compared with those of the country, especially in the city children living in crowded districts, where air and sunlight are insufficient and the mode of living unhealthful. How improper feeding and poor environment are causative in the production of rhachitis is a subject for further speculation and study.

*Gross pathology.*—While all the organs of the body suffer from the effects of rhachitis, the essential lesions are in the bones. The grosser changes require a brief description.

The long bones ordinarily grow in length by the formation of bone in the cartilage between the epiphysis and the shaft; in thickness, by the formation of bone beneath the periosteum; and at the same time the medullary canal grows in size by the absorption of the inner layers of the bone (Holt). In health the seat of greatest activity in growth is at the epiphyses; in rhachitis the most marked changes are found in the same locality. Thus in a rhachitic long bone the epiphyses are enlarged and rounded, and the cartilage between the epiphysis and the shaft is wider and thicker than normal, while the process of ossification is delayed or may be entirely arrested. In the shaft of the bone the outer layers are thickened and soft, and the absorption of the medullary layers is more rapid than normal. The proportion of organic to inorganic matter is much increased, rendering the bones softer and more flexible. The long bones most affected are the ribs and the large bones of the extremities, most frequently those of the leg and forearm.

The flat bones are affected most markedly in the region of the centres of ossification, where a thickening occurs, giving rise to soft and spongy prominences, most often seen in the frontal and parietal region of the cranium.

As the disease subsides after some months, the epiphyses lessen in size, the prominences of the skull decrease, and there is a normal formation of bone. The parts affected often never regain their natural size, and are almost always harder than normal, owing to a process of condensation and contraction in the bony structure.

*Early symptoms.*— The pathognomonic symptom of rhachitis is the development of the bone lesions, but premonitory signs occur which lead to a suspicion of its oncoming.

Perhaps the earliest of these is profuse sweating of the head accompanied by irritability and restlesssness, particularly at night.

Delayed dentition is present in a large proportion of the cases, is a very suggestive symptom, and should always put the practitioner upon the watch for other rhachitic manifestations.

Enfeebled muscular action, again, is a suspicious condition; and delay in holding up the head, in sitting up or walking, is a frequent premonitory symptom. This apparent backwardness is often the most fruitful cause of anxiety to the parents, other signs remaining unnoticed, or thought to be of little importance.

A persistent disturbance of the bowels, either constipasion or diarrhœa, may also herald the approach of the disease.

*Later symptoms. A. Osseous system.* Of the bone lesions, one is first and constant, according to Morse, and that is the "rosary" or beading at the junction of the ribs with their cartilages. This is not present in normal children, and its occurrence justifies the diagnosis of rhachitis. The beading is always greater on the inside than on the outside of the chest wall.

The anterior fontanel normally should be closed by the 18th or 20th month, but in rhachitis usually remains widely

open after that time; the posterior fontanel also occasionally remains open.

The formation of the frontal and parietal bosses previously mentioned gives the head a curiously square appearance and causes an actual increase in its circumference. The parietal eminences are more apt to be enlarged than are the frontal.

Craniotabes may occur. The other symptoms resulting from changes in the osseous system give rise to deformities more or less noticeable and permanent according to their severity.

The chest shows an increase in its antero-posterior diameter caused by the effect of atmospheric pressure upon the yielding ribs. This may be so marked as to form a distinct pigeon breast.

If the entrance of the air to the lungs be impeded by the presence of adenoids or enlarged tonsils, the sternal region may be depressed, causing the funnel chest.

There may be, and very often is, a lateral depression about the chest, corresponding to the insertion of the diaphragm. The contraction of this muscle pulls in the ribs at the point at which it is attached to them, and causes a furrow in the chest wall. This brings about a flaring of the lower costal region which is accentuated by the presence of the normally large liver of infancy.

An almost invariable symptom is the enlargement of the epiphyses of the long bones of the extremities, most marked at the wrists and ankles.

The diaphyses of the long bones also show changes, both bending and torsion, resulting from muscular pull and improper distribution of weight upon the softened bony structure. Bow-legs are the commonest of these deformities, varying from the mild cases where the condition is hardly more than an enlargement of the epiphyses at the ankle, up to the severer ones where walking is almost impossible. Knock-knee is frequent. Deformities of the forearm, espe-

cially an anterior bowing, are not uncommon. In fact, all the long bones of the extremities may assume distortions of all grades of severity.

Other diseases involving changes in the bones of the extremities may here be differentiated.

In scurvy there is an apparent bony enlargment, but one really due to subperiosteal hemorrhage. Further, this occurs about the shaft and not in the epiphyses of the bone. The extreme tenderness accompanying scurvy, the subcutaneous hemorrhages, and the spongy and bleeding gums enable it to be differentiated. Immediate improvement on antiscorbutic diet clears up any doubt as to the diagnosis.

Syphilis is also frequently accompanied by bony changes in the extremities. Here again the epiphysis is not affected, but the enlargement is at the junction of the epiphysis and diaphysis. It is a boggy infiltration over the bones and not an actual enlargement of the bone itself. It may be unilateral and may break down, conditions not found in rickets. The latter bone lesions of hereditary syphilis affect the shaft rather than the extremity of the bone.

In rhachitis there may be occasionally slight tenderness over the enlarged epiphyses. In my experience this has been very uncommon. If sufficient to attract attention and suggest a diagnosis of rheumatism, the absence of local heat and tenderness, the normal temperature and the presence of other rhachitic symptoms render the differential diagnosis simple.

*B. Muscular system.* Rhachitic children are usually fat and may appear at first glance to be well developed. The muscles, however, suffer from malnutrition, as do the other organs of the body. They are weak and flabby, and their action is enfeebled. The delay in holding up the head, in sitting up, and in walking, has already been mentioned.

Another very noticeable characteristic is the enlarged abdomen or "pot-belly." This is due to weakness of the

abdominal muscles, the condition being accentuated by the flaring of the lower costal margin and the atony of the muscular coats of the intestine which results in distension of the bowels. Percussion usually shows marked tympany of the whole abdomen. The absence of fever and free fluid differentiates any abdominal inflammatory condition, such as tubercular peritonitis, which might be suggested.

Weakness of the back muscles may be made manifest by the appearance of a rounded kyphosis involving the lower dorsal and lumbar regions. This is generally accompanied by abnormal flexibility of the spine and usually disappears on recumbency. The reverse may be true, and the persistency and rigidity of the kyphos may suggest caries to such an extent that recumbent treatment for a while is necessary for differentation. The presence of other rhachitic symptoms is of value in the diagnosis and the absence of other symptoms of Pott's disease, such as increased patellar reflexes, psoas contraction and abdominal pain, makes the latter less probable.

Weakness of the muscles of the extremities may be great enough to simulate paralysis, either cerebral spastic or anterior poliomyelitis. The former is ruled out by the absence of mental impairment, of increased reflexes and muscular spasm or contractures; the latter, by the normal surface temperature of rickets, the normal reflexes, the absence of atrophy of any particular region or extremity, and in case of doubt, by the electrical reactions.

*C. Ligaments.* The ligaments show evidence of impaired nutrition in an undue laxity, especially in the lower extremities. Here there may be hyperextension of the kneejoints, but more characteristic are the weak ankles.

Flat foot due to muscular and ligamentous weakness is common, a deformity which is increased by the improper weight distribution resulting from bow-legs and knock-knees.

*D. Respiratory organs.* There are no constant changes in the respiratory system. If the thoracic deformities are marked, especially depressions in the chest wall, the underlying portion of the lungs may be collapsed, with emphysema of the region just anterior to the atelectatic area. The bronchial mucous membrane shows an increased susceptibility to disease, and the respiratory muscles are of diminished power as the result of poor nutrition. These conditions render bronchitis common, especially in the severer cases, and broncho-pneumonia a frequent and dangerous complication.

*E. Gastro-enteric tract.* The intestinal mucous membrane, like the bronchial, is more vulnerable, and gastro-enteric disturbances are therefore common. These may be of all forms and of all grades of severity. There is nothing about them that is characteristic. The atony of the muscular walls of the intestine results in diminished peristalsis, often accompanied by putrefactive changes in the intestinal contents. Thus arises the distension of the bowels already referred to. Either constipation or diarrhœa is usually present, and a normal regularity of the bowels is uncommon. These conditions are fostered by the improper diet of the rhachitic infant.

The liver is seldom increased in size, although its downward displacement by the contraction of the chest often causes it to appear abnormally large.

*F. Spleen.*—The spleen is enlarged in a small proportion of cases, the enlargement being due to simple hyperplasia. There seems to be no relation between the size of the spleen and the severity of the particular case, or the condition of the blood. There is a return to normal size with subsidence of the disease.

*G. Blood.*—The impaired nutrition in rhachitis is accompanied by an anæmia which has no special characteristics. The more marked the rhachitis, the more severe the anæmia

is apt to be. The hæmoglobin is usually relatively low. There may be a leucocytosis.

*H. Nervous system.*—The nervous system of the normal infant is unstable. This condition is aggravated in rhachitis through the defective nutrition of the nervous centres, rendering nervous manifestations early and common. Restlessness at night and irritability have already been noticed. Laryngismus stridulus and tetany frequently occur. Holt states that as a predisposing cause of convulsions in infancy, rhachitis takes the first place. These are due to the quick response of the unduly susceptible nerve centres to relatively slight stimuli.

*I. Glandular system.*—General glandular enlargement is not infrequent, but is not especially characteristic.

Enlargement of the tonsils and the occurrence of adenoids are not uncommon. Their operation in producing thoracic deformity has been mentioned. Their presence gives an added susceptibility to pulmonary complications.

*Diagnosis.*—The premonitory signs of rhachitis, as head-sweating, irritability, restlessness at night, prolonged gastro-enteric disturbances and delayed dentition have been mentioned. These symptoms should excite suspicion. The detection of the rosary and the enlarged epiphyses renders the diagnosis certain. The clinical picture is completed by the appearance of the prominent cranial bosses, the delayed closure of the anterior fontanel, the various thoracic deformities, the pot-belly, deficient muscular action, and as the disease progresses, by the changes in contour of the extremities. The various diseases which might obscure the diagnosis have already been considered.

*Prognosis.*—The prognosis of uncomplicated rhachitis is good. The disease untreated tends to a spontaneous recovery after a duration of several months. With the use of a more general diet after the first year or year and a half, and as the food elements are supplied in better proportion,

there is a gradual improvement in condition. The affected organs slowly regain their normal state unless the lesions have been severe, under which circumstances there may be a varying persistence of the several deformities, even to permanency.

Pulmonary, gastro-enteric, and nervous complications are frequent, and render the prognosis grave. Any acute disease in a rhachitic infant assumes a severe type and is increased in danger.

*Treatment.*—Cheadle says the occurrence of rhachitis is in most cases a grave reflection upon those responsible for the child's diet. Prophylaxis is therefore the most efficient treatment. This comprehends painstaking and accurate feeding, careful hygiene and supervision of methods of living. If, however, prophylaxis has been neglected, then active treatment must be carried out.

Since rhachitis is due largely to the deficiency of fats and proteids in the food, a proper percentage of each must be supplied and improper elements in the diet must be eliminated. Regular feeding must be instituted, and the infant kept under observation that changes in the food may be made when necessary.

Proper amounts of light and air must be secured. City living should be exchanged for life in the country, or better still, life at the seashore. If poverty prevents this, the child may be taken on daily trips to spots where light and air are more abundant. Bathing and massage favor bodily nutrition.

*Drugs.*—The drug treatment of rhachitis is of minor importance. There is no specific for its cure, phosphorus, once recommended so highly, having been practically discarded. While milk and cream afford fat in the best form, it is sometimes expedient to administer it as cod-liver oil in assimilable doses. The anæmia is to be treated by the use of iron.

*Deformities.*—Many deformities may be prevented if their possible occurrences is borne in mind. Creeping, walking, and sitting up may cause bending of the softened bones and should be allowed guardedly until the bones begin to harden. Manipulation will do much to reduce the lesser deformities of the extremities while the bones are flexible. Deformities that are severe and remain in spite of all measures undertaken require orthopedic treatment. Something may be done for the thoracic changes by various exercises, while the distorted bones of the extremities may be corrected by osteotomy at the proper time. The use of apparatus in cases of bow-legs and knock-knee results commonly in annoying failure, and is an unnecessary infliction upon the patient. Osteotomy, when the bones have regained their normal hardness, but not before, is much to be preferred.

ARTICLE LI.

# THE ASSOCIATION OF ANÆMIA WITH CHRONIC ENLARGEMENT OF THE SPLEEN.

By ARTHUR H. WENTWORTH, M.D.
OF BOSTON.

THIS paper is devoted to the consideration of anæmia associated with chronic enlargement of the spleen. Any description of this condition would be incomplete if it were limited to the consideration of cases which occur at any one period of life. I have not included cases in infancy of moderate anæmia with slight enlargement of the spleen because there is no difference of opinion with regard to these cases. Neither does the scope of this article include leukæmia and pseudoleukæmia except in so far as they are to be differentiated from the class of cases which I shall describe. There occurs in infancy and early childhood a severe grade of anæmia associated with marked enlargement of the spleen, the etiology of which has caused a great deal of discussion. In Italy it is called anæmia splenica infettiva, and elsewhere it is known as anæmia infantum pseudoleukæmica. The prototype of this disease in the adult is commonly called anæmia splenica or splenomegalie primitive.

Anæmia infantum pseudoleukæmica was believed at one time to be a primary disease of the blood. Anæmia splenica infettiva and anæmia splenica are still believed by many people to be a primary disease of the spleen. I hope to be

able to show that they are secondary conditions dependent upon a number of causes.

Each of these three titles has its own literature, and it is necessary for this reason to consider them separately. I shall do so in the order in which they were originally described, viz., anæmia splenica of adults; anæmia splenica infettiva of infants; and anæmia infantum pseudoleukæmica.

*Anæmia splenica (splenomegalie primitive.)* The name "anæmia splenica" is one of the many synonyms of pseudoleukæmia. It was employed first by Griesinger to describe cases of severe anæmia associated with chronic enlargement of the spleen. Griesinger believed these cases to be the splenic form of pseudoleukæmia. His observations were clinical.

*In 1866* [1] *Gretsel*, who was an assistant in Griesinger's clinic, reported, under the title of "anæmia splenica," the case of a child with severe anæmia and a large spleen. An autopsy on this case made by Colmheim showed hyperplasia of the normal elements of the spleen; marked increase of young connective tissue in the periphery of the acini of the liver, this connective tissue extended into the acini between the liver cells; increase of interstitial tissue in the kidneys. The lesions did not correspond to those of pseudoleukæmia.

*In 1867* [2] *Müller* reported seven cases which he believed to be cases of *pseudoleukæmia*. He referred to the work which had already been done on pseudoleukæmia and followed this with the description of his cases. They are briefly as follows:—

Case 1. Female. Twenty-six years old; tuberculosis not to be excluded, evidences in both apices that point to the disease; lymph-nodes in the neck enlarged and *painful;* spleen not very large; fever; anæmia; leucocytes not increased in number; died about two years later. Duration of illness three years. No autopsy.

Case 2. Female; fifty-six years old; splenic enlarge-

ment for two years; weakness for one year; shortness of breath; pallor; œdema of legs; fever at times. At the time of observation there was a large spleen; heart and lungs negative; no fever; no increase in number of leucocytes; a gland in the inguinal region the size of an egg; slightly tender; died one year later away from hospital.

Case. 3.—Male, fifty-four years old; enlarged glands and spleen for *six years;* anæmia; weakness; marked loss of flesh; shortness of breath; œdema of legs for two years. Examination showed enormous spleen; slight ascites; leucocytes not increased in number; no fever; died two months later at home.

Case 4.—Male, eighteen years old; nine months before there was jaundice which lasted several months; at time of entrance into hospital slight jaundice; lungs and heart normal; liver rather tender, the border of liver not palpable, liver dulness above at sixth rib mammillary line; spleen large and palpable; leucocytes not increased; seven months later no loss of flesh; slight jaundice; good appetite; no ascites; no increase in number of leucocytes; spleen palpable about two inches below ribs; no fever; liver dulness one inch below ribs, no further report on the case.

Case 5.—Male, fifty-six years old; one year before death enormous spleen; moderate enlargement of the liver; no known cause for either splenic or liver enlargement; attacks of peritonitis in region of spleen; loss of flesh and strength; no increase in number of leucocytes; patient died of general peritonitis from perforation of ulcer in lower part of jejunum. A report of the autopsy says the enormous spleen was rather firm and of *normal* structure and contained numerous hemorrhagic infarcts. No description was given of the other organs and no microscopic examination reported. The nature of the ulcer was not stated. The condition of the liver and lymph-nodes not mentioned.

Case 6.—Male, fifty years old; became weak; easily

tired; rapid emaciation and pallor; no local symptoms; at about same time noticed swelling of the spleen; *gradual swelling* of the cervical, submaxillary, axillary and inguinal lymph-nodes; two years later furunculosis; later obstinate and marked itching of body; brownish discoloration of skin; no fever; epistaxis. Seen at hospital in fourth year of the disease; lungs, heart and liver normal; spleen much enlarged, hard and movable; no increase in number of leucocytes. After two weeks went home and died about five months later.

Case 7.—Male, fifty-six years old; for six years pain at times in left side; splenic enlargement noticed for eight months; slight icterus at times; recent attacks of chilliness, vomiting, constipation, abdominal pain spreading to left shoulder and left leg. At time of examination there was weakness; marked emaciation; pallor; no icterus; furuncles on face; large and hard spleen; liver somewhat enlarged; trace of albumin in urine; leucocytes not increased in number. Died the following month of carbuncle. *Autopsy report:* enormous spleen with adhesions and thickened capsule; firm consistency; pale red-brown on section, trabeculæ and follicles not recognizable; in from five to six places tissue dark red, wedge-shaped, situated at periphery of organ—infarcts.

*Liver* enlarged, surface smooth, capsule thickened; pale red on section, acini distinct; very little blood in large veins. No enlargement of mesenteric and retroperitoneal lymph-nodes. Stomach, intestines, kidneys negative.

Müller examined the organs microscopically and reported as follows: *Spleen*, simple hyperplasia of the normal elements. The connective tissue was especially increased; reticulum thickened noticeably and in its meshes lymph cells but not so numerous as normally; in many places the cells were diminished in number and the connective tissue was correspondingly increased. No evidence of amyloid or of cancer.

It is evident from the above description that no single etiology explains these cases. In two cases tuberculosis cannot be excluded. Case number six corresponds to a case of pseudoleukæmia. Some of the cases are incompletely reported. There were only two autopsies, both of which were reported incompletely.

These cases have been referred to by almost everyone who has written about anæmia splenica, as if they were cases of that disease. As far as I can make out from Müller's article he believed them to be cases of pseudoleukæmia.

In 1871 Wood[3] described a case that he believed to be a third, or *splenic variety* of pseudoleukæmia. The patient was a man thirty years old with anæmia and enlarged spleen; later he became cachectic; there was irregular fever; no increase in the number of leucocytes; somewhat enlarged lymph-nodes in axillæ, neck and groins. The lesions found at autopsy in the spleen were not those of chronic hyperplásia as described by Banti, but resembled more the lesions of pseudoleukæmia. There was no report of the histological examination of the lymph-nodes. It was in all probability a case of pseudoleukæmia in which the most marked lesions were in the spleen. The first systematic work was done by *Banti*[4] *and published in 1883*. Since then a number of men who believe that anæmia splenica is a primary disease of the spleen have written on the subject. Most of the articles are reports of cases. A few of them describe the disease. These descriptions are for the most part repetitions of Banti's work. It will suffice therefore if I give Banti's article more in detail and afterward allude briefly to the articles which have been published since. Banti claims that anæmia splenica is *a primary disease of the spleen* and that it is the *splenic form of pseudoleukæmia*. The characteristics of the disease are said to be a marked primary enlargement of the

spleen without any change in the lymph-nodes; followed by severe and progressive anæmia with marked diminution in the number of red corpuscles and in the hæmoglobin, without any increase in the number of leucocytes. This is followed in a varying length of time by cachexia and death.

Banti's statements are based upon *three* cases that he observed previous to 1883. A brief description of these cases is as follows:

Case 1.—Seventy-two years old: with a chronic progressive anæmia and enlarged spleen. The patient died of pneumonia. The autopsy showed pneumonia; atheromatous changes in the arteries; degeneration of the myocardium; catarrh of the intestines; liver larger than normal; spleen enormously enlarged, capsule thickened and consistency of the spleen increased. There was no evidence of "lymphomatous infiltration" in any of the organs examined. The lymph-nodes were not enlarged; there was a slight increase in the connective tissue of the liver; no lymphoid tissue in the bones.

Case 2.—Eighteen years old; no etiology given. Clinically the patient presented the symptoms of a large spleen and a progressive anæmia. The patient died and an autopsy was not made.

Case 3.—Sixteen years old; no etiology given; the blood showed 3,900,000 red corpuscles and 7,000 leucocytes in a cubic millimetre; the hæmoglobin was 68 per cent. There was ascites; œdema; a large liver(?), and a very large spleen. (The enlargement of the spleen was said to have *preceded* the other symptoms.) There was doubt as to the cause of the ascites, whether due to peritonitis or not, but finally splenectomy was performed for the treatment of the splenic anæmia. The patient died soon after the operation. *At the autopsy* was found chronic peritonitis; liver *small* and granular (weight 1,050 grams), tissue firm and resistant (cirrhosis of liver). The spleen was large; capsule

and septa thickened and fibrous; the follicles nodular, larger than normal and with sclerosis of the walls of the central arteries; the reticulum thickened and fibrous; the pulp contained fewer cells than normal.

Banti believed that the spleen in this case was affected first and later the liver. His reason for believing so was that he could find no etiology for the cirrhosis of the liver.

We have here two autopsies. In one there was *chronic hyperplasia* of the spleen occurring in an old man with chronic changes in the vessels and in the heart's muscle. In the other a marked *chronic hyperplasia* of the spleen, together with *chronic interstitial hepatitis* of marked degree and a chronic peritonitis. In neither of the cases were there any evidences of "lymphomatous infiltration" in the organs, such as one finds in leukæmia and in pseudoleukæmia; whereas some of the usual causes of chronic hyperplasia of the spleen were present in both of the cases.

These lesions of chronic hypherplasia of the spleen are the ones that Banti and others describe as *characteristic* of splenic anæmia. It is important to remember that Banti bases the pathology of the disease splenic anæmia on *these two autopsies* and at the end of his article he shows several drawings of the microscopic appearances of the spleens from these two cases. It certainly does not add to the conclusiveness of his argument that these two cases should have had in various organs and tissues, lesions that frequently are associated with chronic hyperplasia of the spleen as a *secondary* condition.

With regard to the etiology Banti says that rickets, syphilis and tuberculosis may be present in some cases but he denies any causal relationship between splenic anæmia and these diseases. He believes that the changes which take place in the splenic tissue cause disturbances in the splenic functions, the result of which is the production of a toxine that circulates throughout the body and produces

the anæmia and the cachexia. In connection with the pathology of the disease Banti admits that there have been very few post-mortem observations thus far. He says "so far as my own observations go, I am compelled to admit that the lesions in the spleen are *not those of leukæmia or of pseudoleukæmia.*"

Banti repeatedly makes the statement, however, that anæmia splenica is the splenic form of psuedoleukæmia. The only explanation of this contradiction that occurs to me is that he thinks the splenic changes of increased fibrous tissue are similar to the changes that are found at times in the lymph-nodes in the so-called hard form of malignant lymphoma. The following description of the lesions in the liver and spleen is taken from Banti's article:—

"The liver shows some increase in interlobular tissue, containing in its meshes a varying number of lymphoid cells. This change is not marked. The liver cells are normal or somewhat degenerated." He considers the liver changes of no particular importance. "*The spleen* is very large, the capsule is thickened in places and often adherent to neighboring tissues. The color is red-brown on section with whitish-gray streaks and specks more or less numerous. The gray areas are the enlarged follicles and thickened trabeculæ and the septa which are thickened and fibrous. *Microscopically* the follicles are much altered. The central artery is thick and fibrous; the normal follicular tissue is replaced by an irregular thick fibrous network which contains very few lymphoid cells in its meshes. In places the follicles are completely sclerosed, the trabeculæ are thick and fibrous and the spaces between the fibres are so small as to contain only a few cells. In places the trabecular tissue is replaced by dense bands of fibrous tissue with only an occasional cell in the spaces, the veins are dilated and their endothelial cells project into the lumen. There is no pigmentation." "The lesions are (1) atrophy and sclerosis of

the follicles. (2) The normal pulp is replaced by a dense fibrous tissue with few cells." Banti admits that the histological changes in the spleen in anæmia splenica are *not specific*, *i. e.*, not confined to this disease alone, but he sees an analogy in the lesions to those of pseudoleukæmia and the hard form of malignant lymphoma. He says, "it cannot be denied that other morbid conditions are capable of producing similar lesions in the spleen but one must not forget that it rarely is possible to make a diagnosis from the examination of a fragment of one organ. It is necessary to examine the other organs macro and microscopically. Thus in malaria the same appearances may be found in the splenic tissue, but in addition there is pigmentation."

This is an unfortunate argument for him to employ because in both of his cases the changes in the spleen might very well have been *secondary* to lesions in other organs.

Banti believes that the splenic enlargement *precedes* the anæmia. He admits that this is difficult to prove because the patients do not consult a physician as a rule until both conditions are well marked. He refers to the marked enlargement of the spleen at a time when the anæmia is moderate and argues that it must have taken a long time for the spleen to attain such a size and that it did not produce symptoms at first. He refers to Pèan's and Franzolini's cases of splenectomy in support of this statement. I shall refer to splenectomy later and will say a few words about these cases then. Banti divides the disease into three stages. 1, stage of splenic enlargement; 2, stage of progressive anæmia; 3, stage of cachexia. He says that there is nothing characteristic about the blood. It simply shows the changes of a secondary anæmia. Fever, aside from possible complications, is generally observed only in the last months of the illness. It is irregular and intermittent in type; periods of apyrexia of several days' duration are not infrequent. Banti says that the same type of fever is seen in other severe

anæmias such as leukæmia, pseudoleukæmia and pernicious anæmia. The cause of the fever has not yet been discovered. These statements as to the fever do not agree with the statements of some other observers, notably those of Somma, Cardarelli and Fede, who have described the infantile form of anæmia splenica.

The liver enlargement is slight or moderate and is due to congestion and to a slight increase in its connective tissue. Banti says that these changes may be due to the toxine secreted by the spleen which produces the anæmia and other symptoms. Banti's principal reasons for believing anæmia splenica to be a primary disease of the spleen are, 1, the large size of the spleen at a time when the anæmia is not very marked. 2, the enormous size of the spleen, which far exceeds the moderate enlargements that one frequently finds associated with secondary anæmia. 3, the good results obtained by splenectomy. The disease terminates fatally in most cases in which the treatment is hygienic and tonic. Of twenty-one cases of splenectomy performed for various reasons Banti regards four as cases of splenic anæmia. These were the cases of Spencer Wells, Péan, Czerny and Franzolini. Three of the patients recovered and these results confirm Banti in his belief that anæmia splenica is a primary disease of the spleen and that the removal of the spleen is indicated in the early period of the disease before cachexia appears. I have looked up three of the four cases to which Banti refers. They do not convince me that anæmia splenica is a primary disease.

Case 1. (*Spencer Wells.*[5]) Female, thirty-three years old; one brother and two sisters died of tuberculosis. Toward the end of the year 1864 the patient complained of weakness and malaise but the abdominal enlargement did not show itself until April, 1865. (In this case the anæmia and other symptoms appeared to have preceded the splenic enlargement.) In October, 1865, the spleen was

enormous; no œdema or ascites; slight leucocytosis. Splenectomy was performed and the patient died six days later. There was no report of the lesions found in the spleen and no autopsy report.

CASE 2. *(Péan.*[6]*)* Female; six months before the operation evidences of severe anæmia; hæmoptysis; hæmaturia; bloody stools; hæmatemesis; icteric color, etc. Splenectomy was performed and the spleen was found to be very large and *friable*. (There was no report of the histological appearance of the spleen.) Pèan stated that the tissue was so friable that it required the utmost care to pull the spleen down to the abdominal incision. Three months later the patient was well except for slight pallor.

CASE 3. *(Czerny.*[7]*)* The patient had had uterine trouble for several years with menstrual disturbances. The catamenia lasted eight days as a rule. At the time of operation she was anæmic and had a swelling in the left side of the abdomen that was thought to be an enlarged kidney. It was found to be an enlarged spleen and was removed. The weight, minus the blood which it contained, was only 365 grams. The spleen was examined by professor Arnold who reported simple hypertophy. A blood examination was not made until nine months later. At that time the red corpuscles numbered 3,916,000, and the leucocytes 8,900. Attacks of hysteria which had lasted for five years continued the same after the operation.

(I was not able to obtain the journal in which Franzolini's[8] case was reported.) Banti and others claim that one of the strongest arguments in favor of the primary nature of splenic anæmia is to be found in the good results which follow the operation of splenectomy. In this way the focus that produces the toxine is removed. If this be so how shall we account for the good results which follow the removal of enlarged spleens in cases in which the enlargement is unquestionably a secondary condition?

An instructive example of this kind was reported by *Coupland*.[9] This was the case of a woman who was anæmic and who had an enlarged spleen. The diagnosis was made of anæmia splenica and the spleen was removed. The spleen presented the appearances described as characteristic of splenic anæmia, viz., fibrosis of the trabeculae and atrophy of the follicles. The woman improved greatly after the operation, and was stated by Coupland to have passed from an anæmic to a plethoric condition. *Two years* later there was bleeding from dilated veins in the rectum; hæmatamesis; ascites and death. The autopsy showed a *typical syphilitic liver*.

The cases described by Banti are by no means conclusive. In Spencer Wells's case the anæmia was said to have preceded the splenic enlargement. In Czerny's case there was a history of prolonged uterine trouble and long menstrual periods. The spleen was not very large. In Péan's case the spleen was described as so friable that it was removed with great difficulty. This does not suggest the fibrous condition described by Banti as characteristic, and the improvement in the patient's condition had only lasted three months when the case was reported.

With regard to the stage of the disease in which splenectomy should be performed Banti and others advise early operation before the symptoms of grave anæmia have appeared. No doubt the operation should be performed at this time whether for cases of so-called splenic anæmia or for any chronic enlargement of the spleen other than that of leukæmia. The results obtained in such cases ought not to be accepted as proof that such cases are cases of splenic anæmia, because chronic enlargement of the spleen not infrequently occurs without marked symptoms of any kind.

Pels' and Ebsteins' cases have been referred to by West and others as if they were cases of splenic anæmia. There

is nothing in the description of these cases to warrant such an assumption. *Pel*[10] described four cases in 1885–1887, and included them under the title of pseudoleukæmia, because they had similar symptoms. He stated however that he did not believe them to be etiologically similar. He believed one to be a case of lympho-sarcoma; one showed the characteristic lesions of pseudoleukæmia in various organs; one was probably a case of syphilis with lesions in the liver and spleen and in the fourth case there was an old tuberculous process in the lung together with enlarged bronchial, retroperitoneal and mesenteric lymph-nodes. A microscopic examination was reported in but one of the cases. *Ebsteins'*[11] case was probably pseudoleukæmia. He admitted that the lesions in the lymph-nodes, spleen etc., were typical of pseudoleukæmia, but he believed notwithstanding that it was a chronic infectious disease and he called it "chronic relapsing fever," from the character of the fever which was present. He also reported another case that was still under observation in which the spleen became enlarged during the periods of fever and diminished in size in the intervals.

*In 1887 Shore*[12] gave an incomplete description of a case of enlarged spleen. The autopsy report said that the spleen appeared to be normal; the liver was fatty; no blood examination was reported.

*Potain in 1887*[13] described a case which he called "pseudo leucémie splenique." The diagnosis was based upon clinical observation. A man fifty-eight years old with emphysema and an enlarged spleen. The disease was of four years' duration and the patient was under observation at the time. A blood count showed 2,000,000 red corpuscles without augmentation in the number of leucocytes. The hæmoglobin was diminished to one third of the normal. Potain could find no cause for the splenic enlargement and therefore called it a case of splenic pseudoleukæmia.

*Strümpell in 1888–9*[14] described a case which resembled pernicious anæmia. *He admitted this himself* but preferred to use the term "anæmia splenica," to distinguish it from cases of pseudoleukæmia. The spleen during life was enlarged at times, but at the autopsy it was of normal size and showed nothing characteristic of any disease. The bone marrow was red; the liver cells contained fat and there was fatty degeneration of the parenchymatous organs. This case is referred to by West and others as a case of splenic anæmia.

Apropos of the diagnosis in Pels' and Ebsteins' cases, *Askanazy in 1888*[15] described a case which he had observed for three weeks in which there was multiple enlargement of the lymph-nodes associated with elevated temperature. The clinical diagnosis was tuberculosis, but at the autopsy a general enlargement of the lymph-nodes was found which presented the appearances of malignant lymphoma (pseudoleukæmia.) It was only on microscopic examination that lesions of tuberculosis were found. Askanazy emphasizes the necessity of microscopic examination in all cases.

*In 1891 Bruhl*[16] published a long article on splenic anæmia and proposed the name of "splenomégalie primitive" in place of "anæmia splenica." In the literature one finds Bruhl's name quoted equally with that of Banti. This is certainly an injustice to Banti. Bruhl's work is merely a copy of Banti's. Aside from the name "splenomégalie primitive," there is nothing in it of any importance that has not been said by Banti. Bruhl observed one case in which there was no autopsy. The description of the etiology, symptomatology and pathology are copied from Banti. He differs in one particular from Banti; he says "the anæmia may precede the splenic enlargement (the usual form), or the splenic enlargement may occur early, associated with pain in the region." Banti insists that the disease is prim-

ary in the spleen and that the splenic changes occur first. If Bruhl considers it to be a primary disease of the spleen, as he says, then his statements appear to be contradictory.

Bruhl says "the lesions have been considered to be analogous to those of leukæmia and pseudoleukæmia, but Müller and Banti have shown that certain alterations are present that belong to this disease alone." This is an error on his part. Banti said distinctly "it cannot be denied that other morbid conditions are capable of producing similar lesions in the spleen," etc. (I have already quoted this statement of Banti's and need not repeat it at length.) As far as Müller's cases were concerned there was nothing typical of the lesions described by Banti. In one of Müller's cases there was some increase of the connective tissue in the spleen but in a moderate degree.

*In 1892 Debove and Bruhl*[17] published an article on "splenomégalie primitive" and reported clinical observations on a case. They repeat Bruhl's statements in regard to the splenic changes and the occurrence of anæmia and add that there is no correlation between the size of the spleen and the degree of anæmia. They refer to a case of Rendu's in which there was an *increased* number of red corpuscles in the blood. They are not justified in considering this to be a case of "splenomégalie primitive" in the absence of all symptoms except that of the enlarged spleen. In Bruhl's article it is stated to be the splenic form of pseudoleukæmia, but in Debove and Bruhls' article this is denied. In the discussion which followed the reading of Debove and Bruhl's paper.

*Gaucher*[17a] claimed that he had described a case of splenomegelie before Debove and Bruhl. At the autopsy was found chronic hyperplasia of the connective tissue in the spleen and atrophy of the follicles. He showed a cast and microscopic sections of the spleen.

*Degle in 1891*[18] published a case which he called

splenic pseudoleukæmia; a very incomplete *clinical* observation of a patient with an enlarged spleen. His argument appeared to be that it was not a case of leukæmia and was therefore pseudoleukæmia.

*In 1892 Rendu* [19] described a case of "splenomégalie" in a man thirty-seven years old. The symptoms had lasted for six years. The spleen had been enlarged for four years. The man was born in a malarial district but he never had had malaria. *There was no anæmia*, on the contrary a blood examination showed 6,200,000 red corpuscles and 6,000 leucocytes. This case is referred to by Debove and Bruhl, West and others as a case of anæmia splenica yet the only symptom which could suggest the disease is the enlarged spleen. After six years' duration it was certainly time to expect some symptoms of anæmia if it were a case of splenic anæmia.

A somewhat similar case was reported by *Peter in 1893*, [20] under the title of "splenomégalie primitive." There was a history of five years' duration of the splenic enlargement. The *patient was in good health*. The blood examination showed 5,363,000 red corpuscles and 11,780 leucocytes. Such cases call for little comment.

*Williamson in 1893* [21] published a description of two cases of splenic anæmia: one in a boy of 9 years and the other in a man of 21 years. The boy remained under observation for four months and finally died. The autopsy showed the lesion in the spleen described by Banti, viz., marked fibrous changes in trabeculae and follicles and great diminution of cells. There were a certain number of large phagocytic cells containing red corpuscles. The bone marrow was red and showed a lymphoid condition. There was a small amount of pigment in the liver which contained iron. Lymph-nodes not enlarged. The spleen weighed 2 lbs. 7 ounces. The liver weighed 2 lbs. 12½ ounces. The blood examination showed a diminution of leucocytes

which varied from 2,000 to 7,500. The red corpuscles varied from 2,510,000 to 3,540,000. There was an irregular type of fever most of the time for four months. Sometimes a variation of four degrees between the morning and evening temperature. No history of syphilis, malaria or rickets. The boy was said to have been pale for two years. The autopsy showed an old and a recent endocarditis of the mitral valve without enlargement of the heart. There were also small ulcers in the ileum said not to be typhoid or tubercular. Perforation occurred with peritonitis following.

In the other case there was no autopsy. There was practically no fever, 99°. He was under observation for eight weeks. Blood examination showed no increase in leucocytes and no marked diminution of red corpuscles. The number was over three millions. The spleen was enlarged.

These cases do not resemble each other very much clinically.

*In 1894 Sciola and Carta* [22] published the results of a bacteriological examination made in two cases of "splenomégalie primitive" cultures and inoculation experiments gave negative results.

To avoid repetition it may suffice to say that in a number of cases in which bacteriological examinations have been made, the results have been negative so far as any casual relation between the organisms found and the production of the disease was concerned. In some cases the colon bacillus; staphylococcus aureus and streptococcus pyogenes have been found but these organisms certainly stand in no causal relation to the disease.

*In 1896 West* [23] under the title of splenic anæmia described the case of a man 36 years old who for a year had had attacks of epistaxis and had had also a hemorrhage which followed the extraction of a tooth. He had lost flesh and strength during this period. The temperature

was elevated. The spleen was much enlarged and there was marked anæmia. Several blood counts were made, the number of red corpuscles varied between 1,900,000 and 2,226,000. The leucocytes varied between 50,000 and 75,000. Hæmoglobin between 25% and 35%. A blood examination made six days before death showed no leucocytosis and the leucocytes chiefly lymphocytes. There was an irregular remitting type of fever. Death followed the operation of tracheotomy performed for acute œdema of the larynx. West said that the spleen showed a slight increase in the trabecular tissue. The follicles were much diminished in size and badly devolped. The cellular elements were almost entirely lymphocytes with an occasional large cell, containing several nuclei. Iron reaction was negative. There was some increase of the intercellular tissue in the liver; numerous capillary hemorrhages and some round cell infiltration; iron reaction negative. Some of the organs were sent to Prof. Kanthack for examination. The liver weighed 93 ounces, the spleen weighed 76 ounces. Kanthack reported that the spleen was *very soft* and contained one infarct. The examination was made 24 hours after death. There was some incompetence of the aortic valve with a vegetation on the valve the size of a pea. West added a description of the disease together with the lesions in the spleen which Banti stated to be characteristic, viz., marked fibrous changes. He appeared to think that the lesions in his case were similar.

In connection with this case *Kanthack* [24] who was present at the meeting before which the paper was read, said that he wished to make some corrections in the report on the blood and spleen with which his name was connected. He stated that the spleen showed some fibrous change and some of the normal splenic giant cells. He said that the blood should be examined in all cases by differential stains and that this was not done in West's case. It appeared evident

from his remarks that he had some doubt of the correctness of the diagnosis in West's case. The marked leucocytosis, and the description of a very soft spleen do not agree with the lesions described by Banti as characteristic of the disease.

*In 1896 Taylor* [25] described a case without autopsy which he believed to be a case of splenic anæmia. The case was that of a girl 13 years old who for four years had had more or less pain in the left side. A swelling was noticed about the same time, which increased in size. She had been pale for three years. There was a moderate elevation of temperature. The spleen was much enlarged. There was an enlarged gland in the right axilla and one tonsil was enlarged to the median line. Later a small gland was found enlarged in the right side of the neck. It was movable and situated over the anterior border of the mastoid muscle. A soft systolic murmur was present. The heart was not enlarged. The blood at first showed 2,700,000 red corpuscles; no increase in leucocytes; hæmoglobin 35%. Later the red corpuscles numbered 1,160,000 and the leucocytes 46,400. She died three months later. The description of this case is much more like that of pseudoleukæmia than of splenic anæmia.

*In 1895 Hawthorne* [26] described a case of idiopathic enlargement of the spleen with recurrent attacks of purpura in a child of eleven years. The child did not die.

*Stanley in 1895* [27] described two cases of splenic anæmia together with the post mortem findings. In one case there had been for years a marked diminution in the number of red corpuscles and leucocytes. The spleen was enlarged, the capsule thickened; consistency firm; on section there was considerable increase of fibrous tissue and nothing which resembled the lesions of Hodgkins' disease. The liver showed some cirrhosis of recent date; the pancreas was indurated; the bone marrow was red and gelatinous; the

suprarenal capsules showed marked fatty degeneration. In the other case the lesions were the same except that the suprarenal capsules were atrophied.

*In 1896 Koster*[28] described a clinical case of enlarged spleen that recovered under arsenic and oxygen treatment.

In 1896 under the title of "splenomegalia primitiva" *Terrile*[29] described the case of a man twenty-three years old who four years previously had had an enlarged spleen. In 1895 he returned in poor general condition, with weakness and fever. The liver was slightly enlarged; the spleen was palpable low down in the abdomen. The thorax was negative; the superficial lymph-nodes were not enlarged. There was no evidence in the blood of leukæmia or of pseudoleukæmia (?) His condition was too poor at first to warrant splenectomy. Later he developed signs of peritonitis; and pus was removed from the abdominal cavity by aspiration. Laparotomy was performed and an abscess cavity was found which contained *fetid pus*. His general condition improved and he left the hospital promising to return for splenectomy. No explanation was given of the cause of the cause of the abscess. There seems little to warrant the diagnosis of splenic anæmia in this case.

*In 1896 Pinzani*[30] reported a case twenty-two years old with a very large liver and spleen and ascites. He appeared to regard it as a case of *infantile* hypertrophy of the liver and spleen.

*King in 1897*[31] reported a case of enlarged spleen in which the enlargement diminished after a time.

*Stein in 1897*[32] reported a case in which the spleen was very large; the lymph-nodes were not enlarged; the blood was normal—4,500,000 red corpuscles; 13,500 leucocytes.

*Goepel*[33] (Schmidt's Jahrbuch 1898), exhibited before a medical society a boy of eleven years from whom he had removed the spleen fourteen months before for pseudoleu-

kæmia. At the time of the operation the superficial lymph-nodes were not enlarged and the *blood was normal*. No histological report was made of the spleen. There were no especial changes in the blood after the operation.

*In 1898 Marty* [34] reported a case of anæmia with enlargement of the spleen which he called "splenomégalie primitive." The blood was examined by estimating the relative proportion of red and white corpuscles. The report of the case was very incomplete and in no way justified the diagnosis which was made.

*In 1898 Banti* [35] published an article entitled "splenomégalie primitive con anæmia," in which he repeated his views on the nature of the disease —that it is a primary disease of the spleen and the splenic form of pseudoleukæmia. He complained that Bruhl had copied his work without giving him as much credit as he deserved. He referred to his article entitled "Splenomégalie con cirrosi Epatica" [36] and said that he considered this disease to be a primary disease of the spleen similar to "anæmia splenica." He said that he had observed cases with enlarged spleens and anæmia that after a number of years developed atrophic cirrhosis of the liver and that the patients died of the cirrhosis. These cases were not like any that he had heard of. He said that the same lesions were found in the spleen that are found in anæmia splenica. He was not certain whether these were cases of splenic anæmia or not. In one case splenectomy arrested an incipient cirrhosis of the liver. During the operation the surface of the liver was examined and found to be "granular." He stated that the patient recovered. Looking over the record of the case I find that the report of recovery was made nineteen months after the operation. This is not conclusive because Banti states that the disease may last ten years before a fatal termination occurs and he cannot be certain that the cirrhosis was arrested in this case. It seems probable that Banti has described cases

of cirrhosis of the liver associated with enlargement of the spleen. It is stated in text-books on pathology that not infrequently marked enlargement of the spleen occurs in cases of cirrhosis of the liver at an early stage of the cirrhosis. The enlargement in such cases is due to chronic hyperplasia without evidences of chronic passive congestion. The reason for the splenic enlargement in these cases is not clearly understood.

*In 1898 Maragliano*[37] described a case of splenic anæmia in a man twenty years old. There was a history of weakness; fever; sensation of weight in the left hypochondrium which had lasted for four months. The spleen was very much enlarged; the liver slightly enlarged; lymph-nodes slightly enlarged; the blood examination showed 3,800,000 red corpuscles; no increase in the number of leucocytes; evidences of degeneration in the red corpuscles. He agrees with Banti that it is a primary disease of the spleen which later produces the changes in the blood and the cachexia. He believes that the spleen produces a toxine and sees the proof of this in the good results which follow splenectomy. He finds that the serum of the blood in these cases has a marked globucidal action on the red corpuscles. The lesions which he describes in the spleen are those of chronic hyperplasia. As for the action of the blood serum on the corpuscles he furnishes no evidence that the spleen produces the toxic material.

*In 1899 Sippy*[38] published an article on "splenic anæmia" with references to the literature. He said that the clinical and pathological appearances establish the affection as a "distinct morbid entity which is in reality a splenic form of pseudoleukæmia." He believes the appropriate name to be "splenic pseudoleukæmia." The lesions which he refers to, however, are *not* those of pseudoleukæmia. He says "the histological changes constitute a true hyperplasia and present nothing which differentiates it from other forms of

splenic hypertrophy that are not accompanied by progressive anæmia." He refers to the good results of splenectomy as evidence of the splenic origin of the disease. He refers to seven cases of splenectomy, four of which I have referred to in connection with Banti's work. He described a case with autopsy in a man 45 years old.

*In 1900 Osler*[39] reported fifteen cases which he considered to be cases of "primary splenomegaly." This is a great many more cases than anyone else has ever reported. Aside from these the total number of reported cases is said to be under thirty. The description of the cases, of which I shall give a brief summary, is not at all convincing that the symptoms were due to a primary disease of the spleen. In four of the cases there was a history of malaria and in two of the cases a history of syphilis. Only one case was autopsied. It is quite evident that no single etiology will account for these cases.

Case 1. Male; 36 years old; malaria in India some years previously; no syphilis; recurring attacks of hemorrhage from the stomach; enlarged spleen; progressive anæmia; œdema of legs; ascites; death from hematemesis. red corpuscles 2,000,000; no leukæmia; superficial lymp-nodes not enlarged.

Case 2. 11 years old; female; severe hematemesis in ninth year; again in eleventh year; anæmia; swelling of feet; enlarged spleen; one month before observation severe hemorrhage from stomach (mother said that she lost three quarts of blood in thirty-six hours). Red corpuscles 2,250,-000; leucocytes 7,120. Termination not recorded.

Case 3. Male; 35 years; recurring attacks of hematemesis and melœna between 1885 and 1897 *(twelve years); excellent health in the intervals;* no enlargement of lymph-nodes; report of blood examination lost; no leucocytosis; when first seen was only slightly anæmic; death occurred during a hemorrhage; anatomical summary;

chronic hyperplasia of the spleen; only fatty changes in the liver (no further report made).

Case 4. Male; 33 years; no history of malaria or of syphilis; *nearly seven years* ago first attack of hematemesis; since then at intervals of about a year, very severe attacks in which he vomited blood and passed blood in the stools; enlarged spleen; exploratory laparotomy; stomach and duodenum normal; liver smooth, not cirrhotic; removal of spleen; recovery. Four months after last hemorrhage, red corpuscles 3,000,000; leucocytes 2,800; hæmoglobin 25 per cent.; lymph-nodes not enlarged. Patient reported as well one year after splenectomy. (It would be interesting to know whether he continued to be well.)

Case 5. 38 years; male; *hemorrhages for nearly twelve years;* persistently pale for years; no malaria; no syphilis; great enlargement of the spleen; chlorotic type of anæmia; red corpuscles 4,000,000; leucocytes 6,000; hæmogoblin 30 per cent. No further observation reported.

Case 6. Male; 20 years old; lived in malarial district; doubtful attacks previously; hematemesis and melœna in April and in October, 1899; admitted to hospital in 1890. Brown pigmentation of skin; greatly enlarged spleen; liver not enlarged; superficial lymph-nodes not enlarged; red corpuscles 2,187,000, leucocytes 12,497 at entrance. Given Fowler's solution and improved; slight fever. No subsequent history.

Case 7. Male; 40 years old; lived in malarial district; had malaria when a boy; no syphilis; two years loss of weight and pallor; pigmentation of skin; progressive enlargement of spleen; mucous membranes not anæmic; red corpuscles 4,816,000; leucocytes 5,000; hæmoglobin 55 per cent.; liver not much enlarged.

Case 8. Female; 56 years old; recurring attacks of diarrhœa for three years; dates her trouble from this time; noticed swelling in left side at time of first attack of

diarrhœa; no blood or mucus in stools; tumor in left side had increased; hæmoglobin 60 per cent.; red corpuscles 3,605,000; leucocytes 3,000; no evidence in stools of parasites or eggs; remained in hospital six days and discharged improved; red corpuscles 4,300,000; leucocytes 6,000; hæmoglobin 60 per cent.; no fever while under observation.

Case 9. Male; 58 years old; chills and fever when a child; febrile attacks six years before his death in which enlargement of the spleen was noted; anæmia three years later; spleen enlarged and ascites occurred; spleen remained enlarged and two more attacks of ascites occurred that required tapping. Three years before death red corpuscles 4,400,000; leucocytes 5,100. Four months before death red corpuscles 4,788,000; leucocytes 5,200; hæmoglobin 60 per cent. Toward the last the ascites was so extreme as to require tapping. No autopsy reported. Said not to have had cirrhosis of the liver.

Case 10. Male; 39 years old; lived in a malarial district; occasional attacks of chills and fever; chancre, no symptoms; moderate drinker; epilepsy for eighteen years; increasing weakness for past six or eight months; superficial lymph-nodes slightly enlarged in places; spleen greatly enlarged; liver slightly enlarged; red corpuscles 4,128,000; leucocytes 2,800; hæmoglobin 45 per cent. Improved under treatment during two weeks. No further report made. (Patient did not know spleen was enlarged.)

Case 11. Male; 57 years old; alcohol in moderation; obscure abdominal attack seven years before death, thought to be peritonitis at the time; color not good afterwards; seven years later noticed enlarged spleen; red corpuscles 2,500,000; leucocytes normal; hæmoglobin 37 per cent.; slight swelling of feet; pallor; dyspepsia; loss of flesh; very large spleen; liver not enlarged; superficial lymph-nodes not enlarged; slight fever for several months; some

improvement in blood later; red corpuscles 3,328,000; leucocytes 4,000; hæmoglobin 50 per cent. Death followed the operation of lithotomy.

Case 12. Male; 40 years old; dyspepsia for eight years; no history of syphilis or malaria; temperate man; in 1899 had three profuse hemorrhages from stomach at intervals of few days; no pain associated with them; the abdomen gradually swelled and about two months later was tapped and six quarts of fluid withdrawn; spleen was found to be enlarged at this time; liver not enlarged; ankles not swollen; red corpuscles 4,208,000; leucocytes 4,000; hæmoglobin 45 per cent. No further report made on case.

Case 13. Female; 44 years old; three months failing health; loss of appetite; weakness; for six weeks vomiting and diarrhœa; extremely anæmic; enlarged spleen; enlarged liver; lymph-nodes not enlarged. At time of entrance into hospital the number of red corpuscles 1,540,000; leucocytes 3,300; hæmoglobin 23 per cent. Under observation for five weeks, showed great improvement, gained weight and strength; red corpuscles 3,680,000; leucocytes 4,300; hæmoglobin 54 per cent.

Case 14. Male; 35 years old; syphilis at eighteen years, thoroughly treated; married, with three healthy children; five years illness; recurrent attacks of diarrhœa lasting several weeks or months; stools watery, mucus and a little fresh blood; parasites not found in stool; lost forty pounds in few months. Between attacks of diarrhœa gained strength; three attacks of hematuria few months previously; no evidence of malaria; pigmentation of skin; greatly enlarged spleen; liver slightly enlarged; superficial lymph-nodes not enlarged; no fever while under observation; remained in hospital three weeks; improved greatly; spleen appeared to decrease in size somewhat. At first red corpuscles 3,856,000; leucocytes 4,500; hæmoglobin 55 per cent. Two weeks later, red corpuscles 3,692,000; leucocytes 3,500; hæmoglobin 60 per cent.

Case 15. Male; 43 years old; malaria in 1885; no syphilis; not alcoholic; in 1891 vomited large quantity of blood; a few days later a second severe hemorrhage from stomach; noticed lump in abdomen; in 1895 examined and found to have large spleen; in 1899 has remained in good condition; no more hemorrhages; robust and well nourished; slightly pale; large spleen; liver appeared to be slightly reduced in size; red corpuscles 4,270,000; leucocytes 2,500; hæmogoblin 45 per cent.

At the end of his article Osler says, "Doubt has been expressed as to the existence of a separate and distinct disease to which the term splenic anæmia should be given. We do not know whether the anæmia is the result of the enlarged spleen, or whether, as seems more probable, both are secondary to some cause as yet unknown. Provisionally, until we have fuller knowledge, it is useful to group together, as I have done here, cases of idiopathic enlargement of the spleen with anæmia and without lymphatic involvement, and to label the condition splenic anæmia."

De Renzi [40] (abstract in Wien. Med. Presse 1898) says: "Splenic pseudoleukæmia" is a rare and doubtful disease and should not be confounded with "splenic anæmia." In "splenic anæmia" there is never any new growth of lymphoid tissue in the spleen or in other organs, and recovery occurs in some cases."

*West* [41] gives a description of splenic anæmia in Allbutt's System of Medicine, which is taken largely from Bruhl's description of the disease published in 1891. The references to the literature are incomplete and include many doubtful cases, among which may be mentioned Ebstein's, Pel's, Carr's and Strümpell's.

*Stengel in 1897* [42] says of splenic anæmia, "The observations warrant our disregarding entirely the term splenic anæmia. I do not know of the existence of a separate disease that we may dignify with this title or with such names

as "primary splenomegaly," or "idiopathic enlargement of the spleen." The condition of the blood and the characteristics of the spleen do not differ from those met with in well-marked secondary anæmias or splenic enlargement.

*Chvostek*[43] says in his article on splenic enlargement that in many cases of chronic hyperplasia there are no symptoms. The patients in these cases look and feel well. As a rule, however, there are symptoms. These symptoms are due to the general disease that causes the enlargement, or to the large size of the spleen. Symptoms of anæmia are often associated with this condition as yellowish pallor, loss of flesh, weakness, etc.

*In Real — Encyclopædia d. gesammtan Heilkunde*[44] under diseases of the spleen it is stated that "the enlargement of the spleen due to hyperæmia and hyperplasia is not a disease but a symptom which may be due to a great number of causes. Certain symptoms may depend upon the enlargement, such as pain, sense of weight, etc.

Changes in the blood constantly accompany chronic splenic enlargement. These changes are a diminution in the number of red corpuscles and a corresponding diminution in albumen and iron; occasionally an increase in the number of leucocytes." It is not stated that the spleen *produces* the anæmia but that the same cause may produce both the anæmia and the splenic changes.

"Diseases of the spleen are usually secondary. The organ is largely made up of connective tissue with slight metabolism and little tendency to become affected primarily by disease."

*Langhans*[45] in Virchow's Archives says of the splenic form of pseudoleukæmia, "It occurs in rare cases but only in connection with the *soft form of lymphoma*." "In the cases of pseudoleukæmia described as splenic anæmia careful observation would show the participation of at least some of the abdominal glands."

In the edition of *Strümpell's Text Book* for 1897, under Pernicious Anæmia [46] Strümpell says, "The spleen may be enlarged but it is not as a rule very large and there are no noteworthy changes in its structure." These cases of pernicious anæmia, with marked enlargement of the spleen, are frequently termed "anæmia splenica." Strümpell sees "no reason for separating them from the cases of pernicious anæmia without splenic tumor." *Under the diagnosis of leukæmia* [47] he says, "Cases with a gradual progressive anæmia without apparent cause, associated with a chronic splenic tumor, or more often associated with multiple lymph-node enlargement, without increase in the number of leucocytes, are called pseudoleukæmia or anæmia splenica."

"The rarest form of pseudoleukæmia is the splenic. Only a few cases have been observed."

*Ziegler* [48] in his text book on pathology says that so far as known the functions of the spleen consist in the destruction of red corpuscles and the production of lymphocytes in the follicles. Of chronic splenic enlargement he says, "It may occur in a disease of the spleen combined with a pathological condition of the blood, which may show itself either as leukæmia or as simple anæmia. So that authors have differentiated a leukæmia and an aleukæmic (pseudoleukamæic) hyperplasia of the spleen, or splenomegalie." "Large spleens met with in both forms of cirrhosis of the liver may not be due to congestion in some cases because the appearances are not those of congestion and because in these cases there is no evidence of congestion elsewhere."

*Orth* [49] thinks it very probable that there is no distinct splenic type of pseudoleukæmia. The observations are few and doubtful.

### ANÆMIA SPLENICA INFETTIVA DEI BAMBINI (ANÆMIA SPLENICA INFANTILE).

*L. Somma* in 1884 [50] described this disease under the name of Anæmia Splenica Infantile. Etiology unknown;

chronic insidious course; always progressive; very common in first years of life; a morbid entity not dependent upon chronic digestive disorders or other conditions; related to splenic form of pseudoleukæmia might even be called by this name; essential symptoms of the disease are the splenic enlargement and the grave anæmia; the splenic changes produce the anæmia. He considers the splenic changes to be a true hyperplasia. This disease is the prototype of the one described by Banti in adults; three typical symptoms are: 1, waxen color; 2, fever; 3, large spleen. The fever may be absent; other symptoms are secondary. He describes three stages:

*Early stage.* Insidious, not characteristic, malaise, spleen enlarged.

*Second stage.* Large spleen, marked anæmia, usually obstinate and irregular fever, blood of grave anæmia *without* increase in leucocytes, liver sometimes enlarged.

*Third stage.* Cachexia, enormous spleen, obstinate fever, hemorrhages, weakness, gastro-enteric disturbances, no glandular enlargement, almost always a fatal termination, duration not stated.

He describes the following clinical cases:—

Case 1. Child, 5 years; various ailments in infancy; fed on bread and water; advanced degree of cachexia; recurrent fever not yielding to quinine; large spleen; other organs sound; seen later and spleen found to be smaller; much improved. (Never seen again.)

Case 2. 10 months old; artificially fed: cachexia; splenic tumor: other organs said to be negative; mother reported that another child had died of same disease. (Never seen again.)

Case 3. 14 months old; large spleen; anæmic; organs said to be negative; died six months later. (No further observations made.)

Case 4. 15 months old; other children in the family

had rickets; at eight or nine months digestive trouble; recurrent fever; splenic tumor; quinine did no good; two months later anæmia severe; large spleen; improved under tonic treatment; died later; disease lasted eight months. (No further observations.)

CASE 5. 3 months old; gastro-enteric trouble; lost flesh; anæmic; purpuric spots; large spleen; at sixteen months little better; later much better; finally recovered; spleen somewhat enlarged at twenty-six months.

CASE 6. Hospital case; 14 months old; grave anæmia; œdema of feet; slight œdema of face; large spleen; liver little enlarged; purulent discharge from middle ear; blood not examined; became worse and died in convulsions. *Autopsy report:* anæmia of organs; pulmonary congestion; serum in ventricles of brain; spleen firm and large. (Nothing further.)

CASE 7. Hospital case, 2 years old; grave anæmia; petechiæ; spleen very large and firm and tender; blood showed diminished red corpuscles; slight augmentation of leucocytes; great diminution of hæmoglobin; died with severe anæmia.

*Autopsy report:* anæmia of organs; entero-colitis; large spleen congested and firm, capsule thickened (no further report).

CASE 8. Hospital case, 11 months old; still nursing; small frame; grave anæmia; petechiæ; spleen large and firm; bronchial catarrh; convulsive symptoms; died.

*Autopsy report:* anæmia; increased fluid in ventricles of brain; spleen enlarged and congested, not very firm, hemorrhages into it; liver little congested (no further report).

CASE 9. Hospital case, 4 months old; bad feeding; emaciated; prominent abdomen; grave anæmia; fever; enteric disturbance; spleen enlarged; blood showed diminished red corpuscles and diminished hæmoglobin, slight

increase in leucocytes; gastro-enteric disturbance became severe and patient died.

*Autopsy report:* somewhat anæmic organs; sub-acute enteritis; spleen large and congested (no further report.)

Case 10. Hospital case, 9 months old; grave anæmia; petechiæ; epistaxis; no fever; spleen enlarged; abdomen distended with gas; blood showed diminished red corpuscles; microcytes, diminished hæmoglobin; leucocytes not increased; bronchial catarrh; pneumonia and death.

*Autopsy report:* pneumonia both lungs; slight stasis in bases; enormous spleen congested and firm (no further report).

Case 11. 28 months old; (sister died of pulmonary tuberculosis) fever not yielding to quinine; became anæmic; loss of appetite; spleen enlarged; fever continued; child grew somewhat better in the country for a time; then had vomiting and convulsions with irregular respiration later and died (no further report made).

Case 12. 30 months old. Typhoid fever at 18 months; splenic tumor one year later; fever; grew better and fever ceased; then recurred and abdomen enlarged; yellowish-white color of skin; three months later spleen somewhat enlarged; other organs negative; abdomen distended; superficial veins in abdomen distended; fever (no further report).

Case 13. 3 months old; brother said to have died of same disease; mother cachectic; grave anæmia; fainting attacks; no fever; heart sounds feeble; spleen large and firm; liver more enlarged than spleen (no further report).

I have abbreviated these reports somewhat but I have included all that was said about the physical examination and course of the diseases. Such incomplete observations as these do not warrant consideration, and I have repeated them merely to show that the diagnosis of a primary disease of the spleen from such data is inadmissible.

*In 1890 Di Lorenzo*[51] reported four cases with one autopsy. The lesions in the case that was autopsied were an increase of connective tissue in the spleen; sclerosis of the follicles. The lesions in other organs not reported. Microscopic examination of the organs not reported. No blood examination reported. All of the cases were infants and had enlarged spleens and anæmia. One case may have been syphilis; rickets not mentioned; no enlargement of the superficial lymph-nodes.

*In 1889–90 Fede*[52] gave his views on anæmia splenica infettiva. He preferred this name to any other. He doubted if the disease were a form of pseudoleukæmia because he had seen a child recover under tonic and hygienic treatment. He described two classes of cases both with enlarged spleens, *viz.*, cases with and cases without fever. He believed anæmia splenica infettiva to be an infectious disease of infants. He had observed eight cases in one year. No details of the cases were given.

Cardarelli described the disease as pseudoleucæmia splenica (infettiva) dei bambini. He retained the name which he had applied to a case that was observed and autopsied in 1879. In this case the spleen and the liver were the only organs examined. The lesions resembled those found in pseudoleukæmia and not the fibrous tissue increase which is considered to be characteristic of anæmia splenica. It is probable that this was a case of pseudoleukæmia and not anæmia splenica. In this case the lesions in the spleen are described as "marked lymphatic infiltration of sheaths of small arteries; enlargement of follicles and marked hyperplasia of the splenic cells." The incompleteness of the autopsy report renders it impossible to be perfectly certain of the diagnosis. Cardarelli believed the case similar to the cases of "chronic relapsing fever" described by Ebstein. These cases are believed to have been cases of pseudoleukæmia.

*In 1890 Cardarelli* [83] gave a description of the disease of which the following is an abstract: it occurs most frequently between two and four years, also in infancy; no connection with rickets; denies relation with syphilis, but admits that has seen good results follow treatment with mercury in some cases; denies relation to malaria; a noticeable fact is the occurrence of several cases in same family; thinks obstinate diarrhœa one of the early symptoms; blood shows diminution of red corpuscles and hæmoglobin and no increase in leucocytes; glands not enlarged; liver more or less enlarged; more or less anæmia, usually pronounced; appearance is cachectic; petechiæ if disease lasts long.

He describes three forms: *a*, febrile, or acute; *b*, afebrile, or chronic; *c*, chronic, with recurrent fever.

*a*. *Febrile* or acute, three or four to eighteen months duration; never seen cases early in the disease; does not yield to treatment; enlarged spleen; often enormous, but may not be; no enlargement of lymph-nodes; liver more or less enlarged; usually pronounced anæmia, but not always; appearance usually cachectic; loss of flesh; œdema; petechiæ; nose-bleed; weakness; attacks of fever with intermissions.

*b*. *Afebrile* or chronic; same symptoms as in the febrile form, but no fever; spleen often *larger* and more tendency to hemorrhages; duration longer; slight increase in leucocytes in some cases.

*c*. *Chronic, with recurrent fever*. Intervals of apyrexia may last for weeks or for months; spleen grows smaller in apyretic intervals, may become normal size; anæmia also diminishes; says there are like cases described by Ebstein and Pel; very long course may last through childhood to adolescence or even to adult life; prognosis better in these long cases.

Cardarelli has seen thirty-four cases; six times in several members of same family; in twenty-three cases seven recovered, all the others died. Some lived several years with

anæmia and enlarged spleens. *Some still living* (in 1890) *after twelve or fourteen years.*

The article contains nothing but clinical observations. The reports of the examinations are very incomplete and unsatisfactory. From the data furnished it would be impossible to make an accurate diagnosis on any of the cases to which he refers. The only probable explanation seems to me to be that he has reported cases of secondary anæmia associated with enlargement of the spleen due to a variety of causes. There were no post mortem examinations reported in any of the cases except the partial one in 1879.

In 1890, at the first pediatric congress held at Rome, *G. Somma and Fede*[54] read papers on anæmia splenica infettiva dei bambini. These papers appeared in the transactions of the Society in 1891, and were also published in 1890 in the Arch. Italiano de Pediatria.

Somma's paper consists of references to the literature, chiefly Italian, and a description of the clinical manifestations of the disease. This description is the same as Cardarelli's and I need not repeat it. It will suffice to give his conclusions. His references include Gretsel, Pel, Ebstein. These cases, as I have already pointed out, were not cases of anæmia splenica. Somma refers to Di Lorenzo as the first to suggest the term "infectious" ("infettiva") in 1890.

Somma's conclusions are as follows:—

(1) Anæmia splenica infantile, or pseudoleukæmia splenica as it is called by others, is an illness prevalent in infancy, more frequent than generally supposed. Adults are not exempt but it is probable that in these cases the origin dates back to early childhood.

(2) Ignored or unknown until quite recently, it was brought into the domain of modern pathology by Antonio Cardarelli, Luigi Somma and Francesca Fede.

(3) The disease is characterised by severe progressive

anæmia; chronic enlargement of the spleen which shows special characters not found in other diseases.

(4) Clinically it has certain pathognomonic and accidental phenomena. The first are always present, the latter vary.

(5) In its evolution there are usually three stages: early stage, acme, and stage of cachexia.

(6) There are three forms of the disease: *a*, chronic febrile; *b*, chronic afebrile; *c*, chronic with recurrent fever. Any one of the three forms may appear in the beginning of the disease.

(7) The true nature of the disease has not been discovered. He believes it to be due to a pathogenic organism that finds conditions most favorable for its growth in the spleen, whence it finds its way into the circulation and there produces the anatomical and functional changes which make up the clinical picture.

(8) The course is chronic. The duration from eight months to three years. There are not lacking cases in which the term is extended to adult life.

(9) The result is fatal in most cases; in some the course is prolonged to adult life; in rare cases the patients recover.

(10) The prognosis should be guarded. The treatment is chiefly symptomatic. The indication for splenectomy is still a matter of discussion on account of its gravity. When performed it should be done early in the first period of the disease.

Somma had observed twelve cases up to 1891. He never found the glands enlarged. He does not agree with Cardarelli that there is an acute febrile form but believes it to be a chronic febrile. He agrees that there is little or no increase in the number of leucocytes. These statements of Somma's are worthy of notice because of their incorrectness.

*First:* In his conclusion *No. Three* he says that the spleen shows "special characters not found in any other dis-

cases." The lesions which have been described in the spleen are those of *chronic hyperplasia*. It is necessary merely to consult any text book on pathology to learn how varied the conditions are that produce this change in the spleen.

*Second:* In conclusion *No. Seven* he refers to the infectious origin of the disease and its localization in the spleen. There is no evidence to be found in the splenic lesions, the course of the disease, or in the results obtained by bacteriological examination, to warrant such a statement.

*Third:* He says in reference to the enlargement of the spleen that, aside from malaria, if one finds a very large spleen, in 99 cases in 100 it will be a case of anæmia splenica!

Fede's share of the article refers to the etiology, pathological anatomy and pathogenesis. He says that it occurs usually in the first year of life and that males are more often affected than females. He refers to sixty-four collected cases, of which thirty-five occurred between one and eleven months; nineteen between thirteen and twenty-four months; and ten were several years old. Of this number forty-six were males and eighteen were females. He refers to the circumstance noted by Cardarelli that in a number of cases several children in the same family were affected. He admits that rickets has been observed to some extent in some cases, but believes that rickets, syphilis and bad hygienic surroundings only act as favoring elements to assist the specific organism that produces the disease.

Fede says "the only characteristic lesions are found in the spleen, liver and blood. The glands are normal or slightly enlarged. The liver is often enlarged from congestion and from a slight increase in connective tissue together with cloudy swelling. There may be fatty degeneration and atrophy of liver cells in some cases."

"The spleen is especially affected. It is very large and firm; dark red on section; there is hypertrophy of the connective tissue; the pulp is rich in lymphatic cells; the follicles are hyperplastic."

Fede maintains that it is a *special infection* and not pseudoleukæmia. The organism has not been discovered.

He describes the lesions which Armanni found in the liver and spleen in Cardarelli's case. Fede says the signs of pseudoleukæmia were shown in the liver and spleen of this case. Fede refers to Von Jaksch and says that he has described the same disease under the title of "anæmia infantum pseudoleukæmica." The blood changes in anæmia splenica infettiva are diminution in the number of red corpuscles. The number may be between one million and three million in a cubic millimetre; diminished hæmoglobin may vary between twenty-five and forty-five per cent. Contrary to Von Jaksch he never found a marked increase in the number of leucocytes; the eosinophiles are diminished in number.

The remaining references to anæmia splenica infettiva consist for the most part of descriptions of cases. I shall refer to them as briefly as possible.

*In 1885*[55] *Porter* showed two cases of anæmia with enlarged spleens before a medical society. One of the cases had rickets and the other had tuberculosis of the spine.

*In 1891-2*[56] *Carr* reported personal observations on thirty cases of "splenic anæmia" in children. Seven cases were autopsied, and the report states that "microscopic examinations showed the enlargement to be due to simple hypertrophy with some increase in fibrous tissue. The liver was not found distinctly enlarged. There were no noteworthy changes in the other organs."

In fourteen of the cases there was either admitted or probable evidence of congenital syphilis, and seven of these cases died. In twenty-seven cases there was rickets, more or less severe. The blood examinations were incomplete. (If one may judge from the character of the article the writer appears to believe that any chronic enlargement of the spleen justifies the diagnosis of splenic anæmia.)

*In 1892 Gianturco and Pianese*[57] gave a histological and bacteriological report on the organs from a case seen by Fede.

*The Spleen* showed no increase in interstitial tissue; follicles little developed; pulp very rich in blood; reticulum normal; cells of pulp mostly large mononuclear; small mononuclear cells scarce; endothelial cells prominent in veins and blood spaces.

*The Liver* showed increased connective tissue in Glisson's capsule; vessels dilated; some degeneration of liver cells.

Bacteriological examinations and inoculation experiments negative.

*In 1891 Mensi*[58] described a case which he called anæmia splenica infantile. The child was nineteen months old and at autopsy there were evidences of rickets, noma and tuberculosis. No microscopic report of the organs was given. The author believed it to be a primary disease of the spleen and did not even refer to the possibility of the anæmia and splenic enlargement being due to tuberculosis. And yet he described lesions in the spleen that suggest miliary tubercles; "abundant pulp; numerous scattered white points the size of a pin's head surrounded by a red areola."

Another case described by *Mensi in 1892*[59] is referred to by Fink in Schmidt's Jahrbuch. This was a child 16 months old; hereditary tuberculosis; "scrofulous" lesions previously; large liver; palpable spleen; red corpuscles pale and degenerated (no count reported).

*Autopsy report:* marked anæmia; enlarged heart; enlarged spleen and liver; anæmic kidneys.

*In 1892 Mya and Trambusti*[60] reported two cases of anæmia splenica in infants. They regarded anæmia splenica infantile of Henoch; anæmia splenica infettiva; and anæmia infantum pseudoleukæmica as synonymous. They reviewed the lesions found in the spleen and liver by various

observers and called attention to the fact that they were more or less diverse.

Case 1. 15 months old; male; one child in the family died of tuberculosis and two others died of measles and bronchitis; this child had measles followed by attacks of epistaxis which recurred every fifteen or twenty days; child became very pale and spleen enlarged; at one year child weighed about twelve pounds and had rickets; liver slightly enlarged; spleen very large and firm; moderate fever; blood examination showed 1,688,000 red corpuscles; leucocytes 16,000; hæmoglobin 30%; microscopic examination of stained specimens of blood showed the changes described as characteristic of anæmia infantum pseudoleukæmica, viz., nucleated reds; variation in size, etc. Child died after five days' observation.

*Autopsy report*: the lesions in the spleen were similar to those described by Banti in cases of adult splenic anæmia; marked increase in fibrous tissue; follicles atrophied; cells diminished; trabeculæ thick. The lesions in the liver showed increase in the connective tissue in the periphery of lobules; evidences in the cells of a return to embryonic type as described by Luzet.

Case 2. Female; 14 months old; history of fever, cough and digestive disturbance from eleventh month; rickets present: no syphilis or malaria: weight at fourteen months 13½ pounds: moderate enlargement of spleen; liver negative; blood showed 784,000 red corpuscles; 26,849 leucocytes; 20% hæmoglobin; dried and stained blood showed same character as first case.

*Autopsy report:* same lesions as described in first case.

It is worthy of remark that these cases are the only ones among the Italian references in which a careful examination of the blood was reported.

*Hock and Schlesinger*[61] appear to be the only German observers who believe that anæmia splenica infettiva and

anæmia infantum pseudoleukæmica are different diseases. They described a case of splenic anæmia in 1892 in an infant fourteen months old; the child had rickets; anæmia and enlarged spleen; the blood showed 2,100,000 red corpuscles; 11,600 leucocytes; hæmoglobin 35%. There was no autopsy. They differentiate anæmia splenica from anæmia infantum pseudoleukæmica by the clinical appearances; the absence of nucleated red corpuscles; and absence of leucocytosis.

*In 1895 Gossage*[62] reported clinical observations on two cases of anæmia with enlarged spleens. One case three years old with rickets, fontanelle still open. The observations were incomplete.

*In 1896 Mya*[63] reported the case of a child four years old who weighed twenty-seven pounds. It had had frequent attacks of epistaxis and was profoundly anæmic. The blood showed 2,275,000 red corpuscles; leucocytes not increased; hæmoglobin 42%. The spleen was palpable. After treatment with iron the hæmoglobin increased to 75% and the child gained in weight.

*In 1899 Thistle*[64] reported the case of a child with an enlarged spleen and anæmia. The red corpuscles numbered 3,000,000; the leucocytes 20,000. He states that it was not a case of pseudoleukæmia because the lymph nodes were not enlarged.

### ANÆMIA INFANTUM PSEUDOLEUKÆMIA.

This name was used by Von Jaksch to denote a peculiar type of anæmia which occurred in infants. Similar cases had been described previously by Italians under the title of "Anæmia Splenica Infettiva dei bambini." Von Jaksch believed it to be a primary disease of the blood.

At about the same time Hayem described cases in which more careful examinations of the blood had been made. Within the next few years a limited number of cases were

reported by different observers. It was considered, by the majority, to be a primary anæmia. The disease was described as a severe, progressive anæmia of infancy; associated with a marked enlargement of the spleen and with a moderate enlargement of the liver and of the superficial lymph nodes. The blood showed great diminution in the number of red corpuscles and in the hæmoglobin; marked variations were noted in the size and shape of the red corpuscles. Nucleated red corpuscles were abundant, most of them of the normoblast type. Evidences of subdivision could be found in some of the nuclei; there was marked leucocytosis and in some cases the number of eosinophiles was increased.

Most of the cases ended fatally. In a number of instances the patients were observed for a short time only.

The pathological appearances of the organs were reported in but few cases and in these the descriptions were more or less diverse and incomplete. It is evident however that the lesions were *not* those of pseudoleukæmia and one wonders why the name of pseudoleukæmia was selected. Von Jaksch explained that this name was chosen by him to distinguish these cases from cases of leukæmia and from other severe anæmias. It seems an unfortunate choice because it tends to add to the confusion which exists in connection with the disease pseudoleukæmia.

The lesions in the spleen in cases of anæmia infantum pseudoleukæmica appeared to be those of chronic hyperplasia which affected all parts of the organ but in different degrees. The liver showed slight increase in the connective tissue between the acini and in places accumulation of a limited number of lymphoid cells such as one expects to find in connection with interstitial processes. The bone marrow was not examined carefully. At this age the red marrow is normal.

Hayem and Luzet considered the large number of nucleated red corpuscles with evidences of karyokinesis in some of the nuclei to be of great diagnostic value.

I shall describe very briefly the cases of anæmia infantum pseudoleukæmica that have been reported.

*In 1882 Senator*[65] reported two cases eighteen months old; twins; rickets present in both cases; spleens much enlarged; lymph nodes enlarged; a blood count in one case showed 3,266,000 red corpuscles; 43,300 leucocytes. In the other case the blood examination showed 3,860,000 red corpuscles; 23,000 leucocytes.

The first case was under observation for three months without any marked change in condition. The second case died of broncho pneumonia. No autopsy report given.

*In 1888 Von Jaksch*[66] reported the case of a boy fourteen months old with marked enlargement of the spleen; glands in neck, axillæ and groins enlarged and hard; considerable anæmia; blood counts at different times showed variation in red corpuscles between 2,310,000 and 4,500,000; leucocytes 84,000 to 192,000. The liver was somewhat enlarged; fever was present at times; child under observation for six months; toward the last he became œdematous; petechiæ appeared in the skin; and the spleen increased in size still more; death occurred after six months. *Autopsy report* by Professor Eppinger showed typical lesions of leukæmia in the liver, spleen, glands and kidneys.

*In 1890 Von Jaksch*[67] published two more observations. The first one was that of a child eleven months old; grave anæmia; rickets; enlargement of the spleen and lymph nodes; no enlargement of the liver; a blood examination showed 820,000 red corpuscles; 54,000 leucocytes; fifteen per cent. hæmoglobin. *Autopsy report*, spleen large and firm; liver hard; mesenteric lymph nodes enlarged, pale and firm; fatty degeneration of heart; chronic splenic tumor, no trace of leukæmia in the organs.

The second case was that of a child nineteen months old; skin very pale and a trifle œdematous; moderate rickets; general enlargement of lymph nodes; liver not enlarged; spleen enlarged; under observation two and a half months; blood examination at first showed 1,380,000 red corpuscles; 114,500 leucocytes. One year later the blood examination showed 6,043,750 red corpuscles and 50,000 leucocytes. The child was perfectly well. (Luzet refers to this case as doubtful.)

It was upon these three cases that Von Jaksch based the description of anæmia infantum pseudoleukæmica. One case had rickets and chronic hyperplasia of the spleen; another recovered; and the third showed typical lesions of leukæmia in the organs.

*In 1891 Luzet*[68] published a thesis on the anæmias of infancy. Under the title of "anémies pseudo-leucémiques" he described a case observed by Hayem; one which he had observed himself and the three cases of Von Jaksch which I have referred to. Hayem's case was that of an infant of ten months; very anæmic and feeble; slight œdema of legs; no enlargement of lymph nodes; liver somewhat enlarged; spleen very large; no evidence of rickets; blood examination showed 1,356,250 red corpuscles; 33,000 leucocytes; dried and stained preparations of blood showed variations in size; poikilocytosis and evidences of degeneration in the red corpuscles; also numerous nucleated red corpuscles of various types; many of the nuclei undergoing subdivision; nothing characteristic in the leucocytes.

Luzet's case was one year old; digestive disturbances for several months; emaciated; anæmic; no teeth; fontanelle open; marked enlargement of the spleen; lymph nodes not enlarged; liver not enlarged; under observation for four days; blood examination showed 1,498,416 red corpuscles; 15,000 leucocytes.

*Autopsy report:* spleen very large; thickening of the capsule in places; section red; nothing abnormal in reticulum; vessels normal; rudimentary follicles; increased pulp; liver showed evidences of return to embryonal type; dried and stained specimens of blood showed numerous nucleated red corpuscles in many of which the nuclei were undergoing subdivision.

*In 1891 Baginsky* [69] reported five cases very incompletely. *One of the cases* was autopsied and showed chronic hyperplasia of the spleen. The cervical and mesenteric lymph nodes were also enlarged. There was no tuberculosis. No blood examination reported. The clinical symptoms were anæmia; enlarged spleen and liver; enlarged lymph nodes.

*The second case* was that of a child 22 months old; nutrition was good but the appearance was cachectic; there was marked rickets; large firm spleen; enlarged liver; several examinations of the blood were made and showed variations in the number of red corpuscles between 3,995,000 and 4,005,600; the leucocyte count varied between 21,000 and 65,000; hæmoglobin 35 to 39%; a small number of nucleated red corpuscles reported.

*The third case* was that of a boy 11 months old; very much emaciated; marked rickets; very large spleen; liver enlarged; the red corpuscles varied in number at different times between 3,277,980 and 4,350,000; the leucocytes varied in number between 40,000 and 122,222; hæmoglobin 35 to 40%. Dried specimens of blood showed poikilocytes; nulceated red corpuscles in considerable numbers.

*The fourth case* had rickets; anæmia; enlarged spleen; moderate diminution of red corpuscles and hæmoglobin (32%); leucocytes 44,800.

*The fifth case* had enlarged liver and spleen; cervical, axillary and inguinal lymph nodes enlarged; spleen not very large; no blood examination reported.

*In 1891 Holt* [70] reported the case of a child two years old under the title of "pernicious anæmia," but the description resembles the cases that have been reported as anæmia infantum pseudoleukæmica. The child had lived in a malarial district but had not had malaria; the spleen was enlarged; there was a diminution in the number of red corpuscles, and a marked increase in the number of leucocytes; no differential count reported.

*The autopsy* showed chronic changes in the spleen associated with passive congestion; the heart was fatty; there was an old pneumonic process in the lungs.

*In 1892 Tissier* [71] published an article entitled "anémie infantum pseudoleucémique." He believed anæmia infantum pseudoleukæmica and anæmia splenica to be the same disease. His article contained nothing new.

*In 1892 Alt and Weiss* [72] gave a brief report of four cases. Two of the cases had rickets, and a third had syphilis. They stated that the blood in these cases showed the characteristics described by Luzet.

*In 1892 Hock and Schlesinger* [73] reported two clinical observations in infants. Both infants were one year old; both had marked rickets; enlarged lymph nodes; very large spleens; enlarged livers; anæmia; blood examinations showed in one case 2,700,000 red corpuscles; 48,000 leucocytes; 45% hæmoglobin. The other showed 2,900,000–3,100,000 red corpuscles; 45,000–40,000 leucocytes; hæmoglobin 35–42%.

*Koplik in 1892* [74] gave a clinical report of two cases. Both cases had rickets; enlarged spleens; enlarged livers; lymph nodes slightly enlarged in one case. In one case a blood examination showed 2,304,000 red corpuscles; 27,800 leucocytes; 40% hæmoglobin. Dried slides showed numerous nucleated red corpuscles in many of which there were evidences of subdivision in the nuclei.

In the other case only dried specimens were examined. There appeared to be no marked increase in leucocytes.

*In 1892 Monti and Berggrun* [75] described four cases which they had observed and also referred to some of the cases which I have described.

*The first case* was eight months old; emaciated; livid color; severe rickets; chronic pneumonia; liver not enlarged; very large spleen; petechiæ; œdema; trace of albumin in urine; three blood examination as follows:—

| | | | | | |
|---|---|---|---|---|---|
| Red Corpuscles | 2,900,900 | Leucocytes | 45,000 | Hæmoglobin | 42% |
| " | 2,750,000 | " | 48,000 | | |
| " | 2,661,000 | " | 48,714 | | 42% |

A limited number of nucleated red corpuscles.

*The second case* was 19 months old; extremely emaciated; extreme pallor; severe rickets; liver slightly enlarged; spleen very large; enlarged inguinal lymph nodes. The child died after eight days' observation. Autopsy not obtained.

Blood examinations as follows:—

| | | | | | |
|---|---|---|---|---|---|
| Red Corpuscles | 2,422,000 | Leucocytes | 28,686 | Hæmoglobin | 30% |
| " | 2,113,000 | " | 31,600 | " | " |
| " | 2,115,000 | " | 34,500 | " | " |

A limited number of nucleated red corpuscles.

*The third case* was 7½ months old; emaciated; cachectic; severe rickets; enlarged liver; very large spleen; inguinal lymph nodes enlarged.

Blood examinations as follows:—

| | | | | | |
|---|---|---|---|---|---|
| Red Corpuscles | 3,200,000 | Leucocytes | 38,866 | Hæmoglobin | 40% |
| " | 3,210,000 | " | 40,000 | " | " |
| " | 3,248,000 | " | 41,000 | " | " |
| *Last Exam.* | 4,052,000 | " | 18,300 | " | 44% |

Limited number of nucleated red corpuscles. Some improvement noted in general condition.

*The fourth case* was 15 months old; there was extreme emaciation; petechiæ; slight cyanosis; nutritive disturbance

in terminal phalanges; liver not enlarged; spleen much enlarged; inguinal lymph nodes enlarged.

Blood examination as follows:—

Red corpuscles, 2,100,000; leucocytes, 21,000; hæmoglobin 35%; nucleated red corpuscles with mitoses in some of them.

*In 1894, Andeoud*[76] reported the case of an infant eighteen months old; rickets; no fever; lymph nodes slightly enlarged; liver enlarged; spleen very large; incomplete blood examination; there appeared no be no leucocytosis; hæmoglobin 30%; red corpuscles not counted, but were diminished in number.

The autopsy showed hyperplasia of the follicles in the spleen; proliferating lymphoid cells; increased pulp; congested vessels.

Andeoud gives a number of references which include Italian cases of anæmia splenica infettiva.

*Glockner in 1895*[77] published observations on four cases. All of the cases were autopsied; syphilis was not present; rickets and chronic gastroenteric trouble had been present in all. No evidence of malaria or of tuberculosis; lymph nodes not enlarged; spleens enlarged, but not otherwise changed macroscopically; microscopically the capsules and reticulum were thickened, as were the trabeculæ and adventitia of the vessels; there was a striking diminution in the number of red corpuscles, without any evidence in the spleens of their having been destroyed there. The follicles showed no changes; no evidence of hyperplasia in pulp cells; growth of epithelioid cells in the pulp and a slight increase in the endotheleum of the vessels; eosinophiles increased; nucleated red corpuscles diminished. The blood showed nucleated red corpuscles with mitoses in the nuclei of some of them; poikilocytes; number of red corpuscles and hæmoglobin diminished. In two cases there was leucocytosis, in one none, and in one a diminished number of

leucocytes. In two cases there were marked changes in the spleen and relatively slight changes in the blood. There were a number of large mononuclear leucocytes resembling the "markzellen" of Müller. The eosinophiles were not increased.

These four cases are very interesting because among other things they show how little connection exists between the lesions in the spleen and the character of the blood.

*Vickery in 1897*[18] published a clinical observation on a child eighteen months old; anæmia; rickets; enlarged spleen; lymph nodes in neck, axillæ, and groins slightly enlarged; the child died; autopsy not obtained. The blood showed 2,500,000 red corpuscles; 25,000 leucocytes; nucleated red corpuscles were numerous and of various types; myelocytes present. Vickery regarded it as intermediate between pernicious anæmia and leukæmia. There was nothing about the case or the blood that differentiated it from the cases which I have described. Vickery evidently interpreted the blood from the standpoint of the adult and not from that of the infant.

*In 1892 Fischl*[19] stated his conviction that "anæmia splenica infettiva" and "anæmia infantum pseudoleukæmica" were identical, and that both were secondary anæmias. He said: "the fact that dissimilar blood examinations have been reported is no proof to the contrary, because the unstable character of the blood in infancy precludes the possibility of diagnosis by this means alone."

He described a case of severe rickets in which the blood was perfectly characteristic of anæmia infantum pseudoleukæmica. The chief symptom, the marked enlargement of the spleen, was absent. There was only a slight enlargement of the liver and spleen; there was emaciation; waxen color; petechiæ; moderate enlargement of the lymph nodes.

The autopsy showed chronic hyperplasia of the spleen; chronic intestinal catarrh, etc.

The only clinical diagnosis in the absence of the large spleen was rickets with anæmia.

The blood showed in 100 fields, 65 normoblasts; 2 megaloblasts; and 4 free neuclei. In almost one-half of the nucleated corpuscles the nucleus had undergone subdivision. In two there were karyokinetic figures; excentric nucleus frequent.

*In 1894 Fischl* [80] called attention to the strikingly large number of cases which the Italians had described. He emphasized the connection between rickets, syphilis, chronic gastroenteric disturbances and anæmia infantum pseudoleukæmica. In reference to Hayem and Luzet's diagnosis from the examination of the blood, he remarks: "It would indeed be a great advance if such a thing were possible with so little difficulty." He believes that malaria is almost the only disease that can be diagnosticated in this way. He refers to cases that clinically present the picture of anæmia infantum pseudoleukæmica, and in which the relation of red corpuscles to leucocytes and the percentage of hæmoglobin give the typical picture, and yet dried and stained specimens of the blood show nothing that is characteristic. On the other hand, he and later Felsenthal have described cases of rickets with typical changes in the blood and only a slight enlargement of the spleen. He says: "The diverse etiology is not so hard to understand if we regard the manifestations as a result of severe general disturbances." He refers the occurrence of fever to intercurrent diseases.

In a discussion of this paper [81] *Raudnitz* said he believed that he had seen cases of glandular tuberculosis and congenital syphilis that gave the same picture as anæmia infantum pseudoleukæmica.

*Epstein* said that he considered it to be a secondary anæmia, and called attention to the fact that in some cases chronic septic conditions in the new born were followed by

anæmia and splenic enlargement. Also the same condition can depend upon tuberculosis of lymph-nodes and on congenital syphilis; on rickets and on chronic gastroenteric disturbances.

*Von Jaksch* admitted that his observations were made on a limited number of cases and that he had not been in a position since then to observe cases of anæmia in children. So that he had not completed those early observations. He admitted that there was nothing characteristic of the disease in the blood changes, but he believed that the clinical symptoms and blood changes combined were typical. He did not agree that it had anything to do with rickets, syphilis, etc., but still believed it to be a primary disease of the blood in infancy.

When one recalls how incompletely the cases he observed were described, and also his own admission that he had never observed any more cases, it is obvious that his remarks made in 1894 can have no added value by being repeated five years later. The above mentioned cases have been rereferred to repeatedly in the literature as typical cases of anæmia infantum pseudoleukæmia. One sees that almost all of the patients had rickets and that some of them had syphilis. Chronic gastroenteric disturbances were rarely absent from the history.

It has been stated that anæmia infantum pseudoleukæmica was not secondary to rickets because rickets does not cause such severe anæmia. Arguments such as this are valueless as proof. Our knowledge of anæmia is limited to its clinical manifestations in most instances. The clinical examination of the blood affords us no hint of the underlying cause for anæmia in a given case. It seems to me that a critical examination of the cases which I have described should convince anyone that all the evidence tends to show that anæmia infantum pseudoleukæmica is a secondary anæmia.

Most of the observations were made at a time when Ehrlich's method of examination had been in use only for a short time. The control work which has been done in the last seven years has proved conclusively that the importance of blood examination by the usual methods is exceedingly limited in its scope and that it was enormously overestimated at the time when differential diagnoses were made between so many blood conditions. In their zeal to be foremost among the observers men made statements that were based upon too hasty and too few observations. It is certainly suggestive that nothing more has been heard from the men who ten years ago believed anæmia infantum pseudoleukæmica to be a primary disease of the blood. The readiness with which infants' blood reverts to a fœtal type has long been noted. Also the fact that the spleens of infants and children become enlarged more easily than do those of adults. Even in adults rapid changes in the size of the spleen have been observed, due to a varying quantity of blood in the organ.

In 1898 one of the German medical societies prepared seven questions relative to the value of blood examination in early infancy. These questions were submitted to Fischl and Siegert for answer. I shall copy the conclusions which were published in 1898 in the Münch. Med. Wochenschrift.[82] The articles were published subsequently in detail in the Jahrbuch für Kinderheilkende, 1899.

Fischl's conclusions are as follows:—

(1) In early childhood there is no histological picture that is characteristic of this or that form of anæmia, and therefore it is not possible from an examination of blood-slides alone to make a diagnosis of a blood disease. It is possible to say from the blood-slides that anæmia is present, and to determine the degree of anæmia; and that is all.

(2) It is not possible to tell from a blood examination in early childhood which one of the blood-making organs

is affected, because the functions of the blood-making organs that are active in fœtal life continue to be so for several months after birth normally, and especially so under pathological conditions. The blood-making function appears to be undertaken by various organs at this time, and therefore the finding of "characteristic cells" is not reliable.

(3) The occurrence of large numbers of normoblasts; evidences of mitosis in the nuclei; the occurrence of megaloblasts; poikilocytosis and polychromatophilic cells indicates a more or less severe disturbance of the blood-making organs depending upon the degree of abnormality in the cells. The prognosis is therefore more or less grave, but these conditions do not indicate the diagnosis.

(4) Only in typical cases of leukæmia with a chronic course is it possible to make a certain diagnosis postmortem of the blood disease. The antemortem examination of the blood frequently disagrees with the results of postmortem examination.

(Fischl demonstrated the truth of these statements by showing blood-slides and organs.)

Siegert's conclusions were as follows:—

(5) For practical purposes clinically, it is possible to make out the presence of anæmia and its degree from blood-slides.

(6) As a means of localizing the anæmia or its cause the differentiation of the various leucocytes is of no value for diagnosis. It has a certain value for treatment and prognosis, to show the progress the patient is making.

(7) The result of treatment may be followed in this way. Improvement or the reverse is shown by the character of the blood. The abnormalities disappear as the anæmia improves. So that the examination of the blood is of considerable value in this respect.

At the end of the discussion which followed the reading of this paper, Fischl said: "After many years of work on

the blood, one gradually acquires a *certain resignation* toward the results. The more one examines, the more uncertain one becomes of the significance of certain manifestations. This is especially the case in early childhood and infancy, for the reasons which I have stated. One fails to find the more or less sharp distinction to be found in the blood of adults."

Fischl called attention to the fact that "lymphocytosis" is quite frequent in the blood of infants, and especially so after intestinal disturbances.

These statements were made by men who for years have examined the blood of infants. Their observations must be allowed to have more value than the chance observations of men who have never made a study of the subject.

*Stengel* [83] says: "It is quite evident from a study of the etiology, the pathological features and the character of the blood, that anæmia infantum pseudoleukæmica is not an independent disease. On the contrary, it seems to be a type of secondary anæmia which may occur in consequence of a variety of affections, and which owes its peculiar character of the blood to the age of the patient rather than to underlying disease." He also says that a similar affection had been described by Italian writers and styled "anæmia splenica infettiva" before Von Jaksch described his case in 1887. The lesions are those of chronic hyperplasia of the spleen and liver, and do not warrant the assumption that there is a separate pathology.[84]

It is generally admitted that "anæmia splenica infettiva dei bambini" and "anæmia infantum pseudoleukæmica" are identical.

It is interesting to note that in almost all of the cases of "anæmia infantum pseudoleukæmica" more or less severe rickets was present, whereas in the literature on "anæmia splenica infettiva" rickets is scarcely mentioned. In a few instances it was noted as present in a slight degree. The appar-

ent absence of rickets in the Italian cases cannot be due to its having been overlooked, because the Italians go so far as to deny any causal relation between rickets and anæmia splenica infettiva.

It has been stated that the spleen rarely is found enlarged in rickets, and in such cases only moderately. Also that when the spleen is enlarged considerably in rickets, that syphilis is also present. The following references are to articles by Fox and Ball, Kuttner, and Starck, on the connection between rickets and enlarged spleens.

*Kuttner*[85] examined sixty cases of rickets, in forty-four of which he found the spleen enlarged. Thirty-three times slight enlargement; nine times moderate; twice very much enlarged. In fifteen cases the children looked normal. In twenty-three they were in moderately good condition; in seven the condition was poor. Kuttner also refers to collected cases.

*Starck* [86] gives a number of references. In one hundred cases that he examined he found the spleen palpable in sixty-eight. Only three of these cases were over three years of age. He refers to the fact that has been noted repeatedly that infants' and children's spleens enlarge more readily under pathological conditions than do those of adults. Most of Starck's cases showed moderate enlargement. He says that the largest ones were found in the cases of severe anæmia and rickets which have been designated as "anæmia infantum pseudoleukæmica." He refers to autopsies on ninety-three cases in which more than half showed chronic enlargement of the spleen. In addition, he referred to one hundred and forty-eight autopsies, in seventy-seven of which there was chronic enlargement of the spleen and no rickets. So enlargement of the spleen is common in children, aside from rickets. It is associated almost as frequently with chronic gastroenteric diseases and with lung diseases. These enlargements are common in the first year

of life, and persist for long periods. The enlargement of the spleen in rickets is due to chronic hyperplasia. At first there is an increase in the pulp and follicles, and later the growth of connective tissue occurs. This change is not characteristic of rickets, but the same appearances are presented when due to other causes.

*Fox and Ball* [87] reported sixty-three cases of enlarged spleens in children. Rickets could not be excluded in a single case. Syphilis was present in twenty-six cases, and possibly more. The number of autopsies was not stated, but the changes found in the spleen were "hypertrophy with perhaps some excessive formation of fibrous tissue." Nearly all the children were in poor condition, flabby and thin, with more or less severe grade of anæmia. Marked anæmia was not always present, even when the spleen was considerably enlarged. In six cases there was marked anæmia without any leucocytosis. The liver was almost always enlarged, and the superficial lymph nodes frequently were hard and easily detected in the neck, axillæ and groins.

It appears evident, from a study of the literature, that anæmia splenica infantiva and anæmia infantum pseudoleukæmica are identical conditions. A plausible explanation of the apparent differences in the two diseases seems to be that the observations were made at different periods. Most of the Italian work was done between 1880 and 1890. At this time modern methods of blood examination were not generally employed. The attention of the Italians seems to have been directed especially to the symptoms. The only marked difference between cases of anæmia splenica infettiva was the presence or absence of fever. The fever was so variable in its occurrence that the Italians felt obliged to describe three classes of cases, viz.: cases with fever, cases without fever, and cases with recurrent fever.

The work on anæmia infantum pseudoleukæmica was done after 1890. At this time the examination of the

blood absorbed every one's attention; in consequence of which, observation of the clinical course of the disease from day to day was more or less neglected. The relative frequency of its occurrence in Italy, and its rarity elsewhere, can be explained by the fact that, outside of Italy, the diagnosis of anæmia infantum pseudoleukæmica was not made in cases of anæmia with chronic enlargement of the spleen unless the blood changes were characteristic.

## SUMMARY.

(1) The blood changes in cases of so-called anæmia splenica are those of a secondary anæmia. The degree of anæmia varies in different cases.

(2) The degree of cachexia, which has been described in these cases, does not always correspond to the blood changes, which are often moderate rather than severe. The percentage of hæmoglobin frequently is over 50 per cent. In one of Banti's cases it was 68 per cent. The number of red corpuscles is often more than 3,000,000 in a cubic millimetre. It is obvious that the cachexia does not depend upon the diminished number of red corpuscles and the quantity of hæmoglobin. It is not improbable that the cachexia and other symptoms are produced by a chronic intoxication similar to that produced by cancer, tuberculosis, etc., and that the splenic and blood changes are merely two of the results which are thus produced. The source of the intoxication is unknown, and it is very probable that it may come from various sources.

(3) It is not easy to see how fibrous changes in the spleen can produce toxine. Fibrous tissue can interfere materially with the functions of an organ, as in cirrhosis of the liver or in interstitial nephritis. But there is no analogy between these organs and the spleen. Fibrous tissue in itself cannot be considered capable of producing toxic or other substances. It can produce mechanical dis-

turbances by interference with the circulation; or from the increase in size which it may produce in an organ; or by interference with the functions of an organ through injury of its cells. We know that the splenic functions, whatever they may be, are not essential to the life or apparently to the health of an individual, and, so far as is known, after the removal of the spleen the functions of that organ are not performed by other organs. Therefore the mere interference of function from an overgrowth of connective tissue in the spleen could not produce the symptoms described as characteristic of anæmia splenica. The functions of the spleen are in no way analogous to those of the thyroid gland and suprarenal capsules.

(4) If it were possible for fibrous tissue to produce toxic substances, it is difficult to account for the absence of such substances in connection with chronic hyperplasia of the spleen when associated with a variety of well-known causes. In these cases the lesions in the spleen are identical with those described by Banti as characteristic of anæmia splenica.

(5) It has been stated that the splenic alterations are primary and precede all of the other symptoms. This statement requires further observation to confirm it. Bruhl says that the splenic enlargement may come before or after the anæmia. The lesions in the spleen are characteristic of chronic hyperplasia, a condition which is associated with a number of abnormal conditions in various organs and which frequently gives rise to no symptoms, or in other cases causes such symptoms as may be produced by the size and weight of the organ.

(6) The lesions found in the spleen in case of so-called splenic anæmia do not warrant the statement made by Banti and others, that this condition is related in any way to pseudoleukæmia. There is no analogy between the hard glandular form of malignant lymphoma and the lesions of

chronic hyperplasia in the spleen. In addition, it may be said that considerable uncertainty exists as to the nature of the hard form of malignant lymphoma, and its classification is a matter for discussion.

(7) A tendency to generalize from observations made upon one or two cases is to be deplored. In the case of splenic anæmia nothing characteristic of a primary disease has been discovered in any organ or in the blood. The number of cases, small as it is, unquestionably shows a diverse etiology. The investigations have been very incomplete, and most of the statements depend upon clinical observations. No better proof of a varied etiology is needed than is to be found in Banti's own cases.

(8) The evidence is conclusive that anæmia infantum pseudoleukæmica is a *secondary anæmia*, and that it owes its peculiar symptoms and blood changes to the occurrence of severe anæmia at an early age.

(9) There is little doubt that anæmia infantum pseudoleukæmica and anæmia splenica infettiva are indentical conditions, and there is even less proof that anæmia splenica infettiva is a primary disease of the spleen than that anæmia infantum pseudoleukæmica is a primary disease of the blood.

(10) There is no apparent connection between the character of the blood and the splenic changes in infancy. Cases with identical lesions in the spleen, viz., chronic hyperplasia, show varying degrees of anæmia. At times there are marked changes in the blood, and at others the changes are very slight. The degree of leucocytosis varies in the same way. In some cases it is considerable, and in other cases the number of leucocytes is normal or even diminished.

(11) These differences cannot be explained by the duration of the condition, in many of the cases. All that one is justified in concluding about these secondary anæmias

of infancy is that in some cases there occurs a chronic hyperplasia of the spleen and in others the spleen is not altered.

(12) The names anæmia splenica, primary splenomegaly, anæmia splenica infettiva, and anæmia infantum pseudoleukæmica, are objectionable because they are misleading. Anæmia splenica has been used for many years as one of the synonyms of pseudoleukæmia, and should not be used to describe conditions that are in no way related to pseudoleukæmia. No evidence has been furnished that justifies the use of the word "primary" in connection with splenomegaly. The same may be said of the word "infettiva" (infectious) in connection with the infantile form of splenic anæmia. Anæmia infantum pseudoleukæmica is a secondary anæmia of infancy, and in no way related to pseudoleukæmia.

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## DISCUSSION.

Dr. H. F. Hewes, of Boston: I think that most men who have had much experience with these anæmias of infancy will agree with Dr. Wentworth that they, as well as the so-called splenic anæmia of adults which he classes with them, are forms of secondary anæmia. The connection of the affection of the spleen with the anæmia in these cases has been a matter of speculation to students of the subject. Some have held that the splenic affection had a causative influence upon the anæmia. This opinion is based principally upon the fact that several cases of splenic anæmia have improved after extirpation of the spleen.

Following the experiments upon animals, which it is claimed indicate that the spleen normally has a marked influence upon the separation of hæmoglobin from the red corpuscles, the hypothesis is put forth that this influence is increased in the splenic hyperplasia of the splenic anæmias. We must, however, await more evidence before we can accede to the spleen a causative rôle in these conditions. It is probable that the affection of the spleen, in these anæmias, is a condition secondary to the same cause which produces the anæmia.

ARTICLE LII.

# CRETINISM.

By CHARLES S. MILLET, M.D.,
of Brockton.

Osler defines cretinism as "a chronic disease of nutrition due to loss or impairment of function of the thyroid gland. It causes a retardation of development of the central nervous system, leading to a retention of an infantile state, and to an extraordinary disproportion between the different parts of the body."

In its endemic form, this disease has been known to exist since the Christian Era began; Strabo, Pliny the Elder and Juvenal all speak of goitre—the handmaid of cretinism—as being prevalent among the inhabitants of the Alps. But it is only about twenty-five years since sporadic cretinism was so carefully described by Sir William Gull, that his name is sometimes attached to the adult form of the disease. Following him, Ord, Kocher and the Reverdins did much; but the physiologists—particularly Schiff, Horsley and Von Eiselberg—made possible the successful treatment of myxœdema and cretinism by their scientific work in vivisection.

The extent to which this disease is endemic in certain parts of Europe is astonishing, and it has attracted so much attention that diligent and persevering inquiry has been made by distinguished scientific and medical men; the subject has also been studied by commissioners ap-

pointed by the governments of Sardinia, France, Austria and Switzerland.

In the eastern part of France there are as many as thirty-two cretins per thousand, and one hundred and eleven cases of goitre per thousand. In the whole of France, over 125,000 cretins and idiots are believed to exist. On the other side of the Alps—in Switzerland and in Austria—the number is fully as great. Contrast this statement with the fact that less than one hundred cases of the sporadic disease are all that have been reported in North America!

The knowledge which we possess of the function of the thyroid gland is of comparatively recent date. That it had any important work to perform, was considered very unlikely; some thought that it was simply a pad to protect the trachea and fill out the contour of the neck; and others thought that it acted as a kind of safety-valve to the vessels feeding the brain. The importance of the organ to life and death was first clearly demonstrated by Schiff in 1884, when he found that the removal of the thyroid gland in dogs was almost invariably followed by profound illness and death.

Further experiments on other animals brought to light the fact that the accessory bodies, which sometimes number as many as four, differ both in structure and function from the gland itself. The physiological difference is yet to be determined, but it has been settled beyond dispute that the removal of the thyroid gland, without the extirpation of the parathyroids—while it produces cretinoid symptoms—does not cause death, more or less suddenly, as is the case when the latter are also taken out.

The endemic form generally occurs in mountainous regions, and in the valleys between the mountains. The idea has long existed that the drinking-water in these places alone contained the organic poison, but at the present time it is believed to be also air-borne.

So-called "goitre springs" are not uncommon in Germany, and Klebs found in one of them an infusoria which he thought caused goitre and endemic cretinism; but other observers have not been able to substantiate his experiments. All agree, however, that the disease in question must have a bacterial origin.

The only essential point wherein the endemic differs from the sporadic type, is that a goitre is much more apt to be present in the former.

Myxœdema—as is well known—is the name given to the same disease in adults, and exophthalmic goitre is their antonym. Several cases are on record where the symptoms of the latter disease have been followed by, or have changed into, those of the cretinoid state; and when the thyroid gland has been removed in an attempt to cure Graves' disease, myxœdema has occasionally been the result.

The disease may vary much in intensity, but even in the slight or early form, diagnosis is never difficult if all the symptoms of abnormal development are carefully considered. Cretinism is quite unusual, in that the physical signs are far more noticeable than the symptoms. It may occur in utero, but generally the first signs of trouble are not visible until the child is five or six months of age, and more often he is three or four years old before it manifests itself.

When the symptoms first become prominent, it is noticed that the child does not grow so rapidly as it should, that it is not so bright mentally, and that its tongue is too large for its mouth and lolls out between the teeth; the skin over the whole body begins to appear thick, swollen, dry and scaly instead of soft and pink; but it does not pit on pressure, or have the glossy, lardaceous appearance that is seen in Bright's disease. Virchow expressed it quite tersely when he said the skin was too big for the body; it

certainly appears so, for it is prone to hang in folds, and even the forehead is sometimes pendulent, and the chin dewlapped. The hair is thin and coarse, and in older children it frequently is not present in the axillæ or over the pubes. The child's face has a decidedly aged appearance, and the eyelids are often swollen so that only a narrow slit shows where the eyes are located. The nose is depressed between the eyes, and the alæ nasi are thick, thus making it seem still more flat, from the great width of the nostrils exteriorly. The ears are also thickened and stand out from the head. The teeth are irregular, ragged and decayed; they are very late in coming, and the second set often never comes, or, if present, the teeth are abnormally large and deformed. The abdomen protrudes; there is generally an umbilical hernia. The back is arched, and curvature of the spine is also frequently a result.

The growth of the child is stunted on account of the limbs being abnormally short, and the hands and feet are undeveloped; the fingers and toes are immobile, and stand apart as a result of the morbid condition of the skin. The nails are short, brittle and striated, and dull in color. The child is distinctly pale, because anæmia is always present, although the cheeks may be slightly reddened. The fontanelles commonly do not close for a long time. The muscles are weak; the head droops—sometimes so much that the chin rests on the chest.

Goitre, which is so common in the endemic disease, is only occasionally present in the sporadic, and as a rule the thyroid body cannot be palpated. Above the collar bones irregular masses of fat may frequently be felt; also upon the arches of the feet, the backs of the hands and in the abdominal walls.

The mental aptitude is even more feeble than the physical condition. The child sits about, not at all interested in his surroundings, and frequently does not even recognize

his parents; if he walks, it is only slowly, with what the Germans call a "bear gait," and this is caused by the lack of coördination from paresis, producing stiffness of the knees. There is, however, no paralysis, and the reflexes are present. The innocent victim exhibits no desires unless, perhaps, for food, for which there may be a voracious appetite. Constipation is usually present. Young children must be fedalmost wholly upon semisolids, for the mucous membrane in all these cases is generally in the same swollen condition as the skin.

Memory is very deficient, and speech is slow, thick and hoarse, and sometimes wanting. The child is evidently idiotic, but the aspect is entirely different from idiocy produced by other causes—such as cerebellar sclerosis or epilepsy. The cretin is not quick-tempered, and does not cry easily; on the contrary he laughs and smiles readily. The child never sweats, nor do the sebaceous glands secrete anything; but drooling is constant and profuse, and there is a secretion of tears.

A very striking objective symptom is the temperature, which is invariably below normal; most frequently it is 97°, or even lower.

In boys the testicles are small and atrophied, and often only one descends. The heart, lungs, liver, spleen and other internal organs are not usually affected.

To sum up,—the organs which are affected by this peculiar disease are the brain, skin, mucous membrane, bones, generative organs and blood; and all of them to about the same degree, although in many cases the body is apparently more diseased than the brain; yet when treated the nervous system shows the least improvement. The severity of the symptoms is of course somewhat dependent upon the length of time the disease has lasted when seen, and upon the age when it began, but mainly upon the degree of impairment of the function of the thyroid gland.

Cretinism may be confounded with fetal rickets, idiocy and infantilism. The children that suffer from congenital rickets are dwarfs, but they do not show changes in the skin, nor are they degenerates mentally. Although the fontanelles remain open for a long time in both diseases, yet in rachitis that began in utero there is apt to be no ossification of the sutures for a long period, wide spaces of soft tissue occurring instead. Of course the cardinal symptoms of rickets are also present to guide us. Idiotic children, not cretins, do not suffer from retardation of growth; their heads are often very small, and the fontanelles close prematurely. Again, the skin and facial aspect are enough to make a differential diagnosis, except in the Mongolian type of idiocy, which resembles cretinism to a marked degree, so much so that if I had such a case I should be inclined to try the effect of the thyroid treatment. But mentally the Mongolian idiots are vivacious and mischievous—never apathetic.

Cases of infantilism that are fat, and especially if they have adenoids, might easily be called cretins, but here again the condition of the skin renders a faulty diagnosis very unusual.

Early and slight forms of the sporadic cretinism are often overlooked; the mental apathy, muscular weakness, stoppage of growth, dryness of skin, together with subnormal temperature, if grouped together, no matter to how slight a degree the individual symptoms are present, are enough to justify an experimental course of treatment with the sheep's thyroid.

In the disease under consideration, when autopsies have been made, the gland has sometimes been found wanting; sometimes atrophied with marked increase of the connective tissue, and degenerative changes in the cells and their nuclei; and sometimes a goitrous condition is present, and this is probably in the nature of a compensatory hypertrophy.

The morbid changes in the skin and mucous membranes are due to the infiltration of a gelatinous substance which penetrates the connective tissue; this produces great compression upon the hair follicles, sweat and sebaceous glands, —which accounts for the loss of hair and the absence of sweating.

The cranial bones are thick; the brain small, with coarse convolutions; the long bones are shortened, and on account of a peculiar crook, their extremities are apparently—although not really—enlarged. There are no visceral lesions peculiar to the disease.

Previous to the experiments of Murray and McKenzie, two English physicians, little or nothing had been done to alleviate this horrible disease. After the Germans and some of his English confreres had failed to produce more than temporary beneficial effects by implantation of the thyroid gland of sheep, Dr. Murray, in 1892, showed that hypodermatic injections of an extract of the gland caused a diminution of all the symptoms; and later Dr. McKenzie fed his patients the same thing by the mouth, and proved it to be equally efficacious when given that way.

The thyroid gland is best given in a powder, since it dissolves rapidly upon the tongue, and none is lost even if the patient is unruly. It is best to begin with half a grain, three times a day, and gradually increase it. Whenever the temperature goes above 100°, or the child becomes very nervous and does not sleep, the remedy must be diminished, or stopped for a while. Later, when the nutritive changes are fully reestablished, a very moderate dose once or twice a week may be sufficient.

I take great pleasure in presenting two cases for observation. This boy is a typical cretin; he is now 9½ years of age, and this photograph was taken when he was 18 months old (and he had to be pinned to the chair, not being able at that time to sit alone). His mother first noticed,

when he was less than a year old, that he could not sit up as her other children had done at that age; also that he was listless; his teeth did not appear till he was nearly four years old, and as yet he has but two of his permanent set. I saw him first four years ago; at that time he could not walk, but could stand up by things.

With the exception of the supraclaricular deposits of fat, he had all the symptoms. He measured thirty inches in height. Under the constant daily use of Armour's dessicated thyroids he has grown fifteen inches in four years. He does not yet put words together, and memory is strangely lacking; his mother says that in going from one room to another, he will forget where he had left any article that he was carrying for her. Although he has made marvellous advances, he still presents most of the signs of the disease.

The other patient is myxœdematous. She is thirty-two years of age, and had none of this trouble until ten years ago. Four years ago she took the powder for some time with decided improvement; but it was neglected, and she now presents all the symptoms necessary for a diagnosis. You will undoubtedly notice the Mongolian aspect, with the bloated face. Let me call your attention also to the teeth, the neck, the hands and the lower limbs. On one leg she has an ulcer, and varicose veins. She menstruates only semioccasionally. Her sister tells me that she had two attacks, during the past winter, of acute swelling of the vocal cords—a condition to which she is probably prone on account of the œdematous mucous membranes.

## DISCUSSION.

Dr. C. W. TOWNSEND, of Boston: The subject has been so admirably covered by Dr. Millet, that there is very little to be added. The history of cretinism illustrates very well the fact that we are liable to overlook obvious signs and symptoms until our attention has been called to them. Thus, in 1893 Osler was able to collect only eleven cases of cretinism in America, and in that paper he mentions the use of thyroid extract as of probable benefit. In 1897 he published sixty cases, and since that time the number of published and of observed cases has increased very much. Now this does not mean that cretinism has increased in this alarming ratio, but simply that our attention having been called to these cases by the remarkable effect of treatment, we have been more critical in our diagnosis, and have recognized cases which were formerly overlooked, and classed as idiots. It is, I think, very important to recognize these cases early, as the results of treatment on the mental development is much more satisfactory in the very young. One of my own cases goes back to the period before the use of thyroid extract and was reported at the meeting of the American Pediatric Society in 1892. At that time Dr. Osler stated that with the exception of references so ancient in Massachusetts that he could not get at them, there was no American literature on Cretinism.

Another case out of the common that I reported in 1896, was a woman 36 years old, a typical cretin, only 3 feet 7½ inches tall, who was delivered by Cæsarean section of a seven months child. The child shown here by Dr. Millet reminds me of another case, when, besides the mental and general physical development, the eruption of teeth was most astonishing on the administration of thyroid extract. The child was four years old and toothless, but in the first four months of treatment it cut seven teeth, and in the next two months five more. We are certainly indebted to Dr. Millet for his paper.

# The Massachusetts Medical Society.

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PROCEEDINGS.

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# The Massachusetts Medical Society.

## PROCEEDINGS OF THE COUNCILLORS.

### October 5, 1898.

A Stated Meeting of the Councillors was held in the hall of the Medical Library Association, No. 19 Boylston Place, Boston, on Wednesday, October 5, 1898, at 11 o'clock, A.M.

The President, Dr. Edwin B. Harvey, in the chair.

The following Councillors were present:

*Barnstable.*
R. H. Faunce.

*Bristol North.*
F. A. Hubbard.

*Bristol South.*
G. De N. Hough,
J. H. Jackson.

*Essex North.*
I. J. Clarke,
F. E. Sweetsir.

*Essex South.*
C. A. Carlton,
H. W. Newhall.

*Hampden.*
G. E. Fuller.

*Middlesex East.*
M. A. Cummings,
S. W. Kelley.

*Middlesex North.*
W. G. Eaton,
J. B. Field,
J. A. Gage,
W. J. Sleeper.

*Middlesex South.*
R. E. Brown,
J. E. Cleaves,
C. H. Cook,
J. G. Dearborn,
A. G. Griffin,
E. D. Hooker,
J. T. G. Nichols,
G. C. Pierce,
E. H. Stevens,
L. R. Stone,
H. P. Walcott,
A. C. Webber,
J. W. Willis.

*Norfolk.*
F. A. Bragg,
J. S. Brownrigg,
C. A. Cheever,
H. M. Cutts,
D. G. Eldridge,
F. P. Emerson,
W. Ingalls,
G. H. Ingalls,
E. G. Morse,
H. R. Stedman,
C. F. Withington.

*Norfolk South.*
O. H. Howe,
J. H. Robbins.

*Plymouth.*
H. W. Dudley,
F. G. Wheatley.

*Suffolk.*
J. B. Ayer,
B. F. Campbell,
D. W. Cheever,
E. G. Cutler,
T. W. Fisher,

R. H. Fitz,
M. F. Gavin,
G. W. Gay,
C. M. Green,
W. H. H. Hastings,
J. Homans,
F. I. Knight,
A. H. Nichols,
J. J. Putnam,
J. M. Putnam,
M. H. Richardson,
W. L. Richardson,
T. M. Rotch,
G. H. M. Rowe,
G. B. Shattuck,
A. M. Sumner,
C. W. Swan,
J. B. Swift,
G. G. Tarbell,
H. F. Vickery,
E. N. Whittier.

*Worcester.*

W. P. Bowers,
E. B. Harvey,
S. P. Holbrook,
E. W. Norwood,
H. M. Quinby,
L. Wheeler.

*Worcester North.*

L. G. Chandler,
E. J. Cutter,
B. H. Hartwell.

Total, 78.

The record of the previous meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

*Vermont.*—Drs. N. P. Wood, of Northfield; W. H. Pierce, of Greenfield.

*New York.*—Drs. H. Bushnell, of North Adams; E. R. Wheeler, of Spencer.

*New York State Medical Association.*—Drs. J. F. A. Adams, of Pittsfield; H. W. Van Allen, of Springfield.

The Committee on Membership and Finance reported through Dr. Draper. In accordance with their recommendation the following were allowed to resign:

Allen Burdick, of Dorchester.
Charles P. F. Joyce, of Dorchester.
Charles W. Page, of Middletown, Conn.
William T. Parker, of Groveland.
Columbus S. Scofield, of Richfield, Vt.
Mary A. Wattles-Faunce, of Fort Collins, Col.

Also the following were allowed to retire:

William H. Hull, of Holbrook.
J. Henry Robinson, of Southboro'.

Also the following was deprived of the privileges of membership under By-Law VIII.:

Henry B. Stoddard, of Newtonville.

The Committee on Publications reported on the question referred to it at the last meeting, of the advisability of sup-

plying the Boston Medical and Surgical Journal to the members of the Society, that such a step would be inexpedient, inasmuch as it would require a not inconsiderable increase in the annual assessment.

*Voted*, To adopt the conclusion of the Committee.

Adjourned at 11.25 A.M.

FRANCIS W. GOSS,
*Recording Secretary.*

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FEBRUARY 1, 1899.

A STATED MEETING of the Councillors was held in the hall of the Medical Library Association, No. 19 Boylston Place, Boston, on Wednesday, February 1, 1899, at 11 o'clock A.M.

The President, Dr. EDWIN B. HARVEY, in the chair.

The following Councillors were present:

*Berkshire.*
O. J. Brown.

*Bristol South.*
G. De N. Hough.

*Essex North.*
F. W. Anthony,
C. G. Carleton,
I. J. Clarke.

*Essex South.*
C. A. Ahearne,
C. A. Carlton,
H. W. Newhall,
S. W. Torrey.

*Franklin.*
W. H. Pierce.

*Middlesex East.*
A. H. Cowdrey,
M. A. Cummings,
S. W. Kelley.

*Middlesex North.*
W. G. Eaton,
J. B. Field,
J. A. Gage,
W. J. Sleeper,
C. A. Viles.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
R. E. Brown,
J. E. Cleaves,
J. G. Dearborn,
E. G. Hoitt,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
L. R. Stone,
H. P. Walcott,
J. W. Willis,
M. Wyman.

*Norfolk.*
F. A. Bragg,
J. S. Brownrigg,
C. A. Cheever,
F. P. Emerson,
H. R. Hitchcock,
G. H. Ingalls,
E. G. Morse,
H. R. Stedman,
L. F. Vickery.

*Norfolk South.*
O. H. Howe.

*Plymouth.*
N. K. Noyes,
F. G. Wheatley.

*Suffolk.*
J. B. Ayer,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
E. G. Cutler,
F. W. Draper,
T. W. Fisher,
C. F. Folsom,
M. F. Gavin,
G. W. Gay,
W. H. H. Hastings,
F. I. Knight,
A. H. Nichols,
C. B. Porter,
A. Post,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
F. C. Shattuck,
A. M. Sumner,
G. G. Tarbell,
H. F. Vickery,
J. C. White,
E. N. Whittier.

*Worcester.*
W. P. Bowers,
E. B. Harvey,
S. P. Holbrook,
J. O. Marble,
H. M. Quinby,
L. Wheeler.

*Worcester North.*
L. G. Chandler,
E. J. Cutter,
B. H. Hartwell,
F. H. Thompson.

Total, 78.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

*Maine.*—Drs. F. W. Anthony, of Haverhill; J. J. Sewall, of Roxbury.

*New Hampshire.*—Drs. G. W. Gay, of Boston; B. H. Hartwell, of Ayer.

*Rhode Island.*—Drs. W. L. Johnson, of Uxbridge; A. C. Getchell, of Worcester.

*Connecticut.*—Drs. T. F. Breck, L. A. Préfontaine, of Springfield.

*New Jersey.*—Drs. J. H. Abbott, of Fall River; J. T. Bullard, of New Bedford.

The following Committee was appointed:

*To Audit the Treasurer's Accounts.*—Drs. F. G. Morrill, H. M. Cutts.

The Committee on Membership and Finance reported through Dr. Draper. In accordance with their recommendation it was voted that the following be allowed to resign:

William C. Billings, of New York, N. Y.
Joseph A. Capps, of Chicago, Ill.
Charles F. Foye, of Haverhill.
Erastus E. Maryott, ofMillbrook, N. Y.
John W. Perkins, of Kansas City, Mo.
Herbert L. Smith, of Nashua, N. H.
William S. Thayer, of Baltimore, Md.
Jerome H. Waterman, of New York, N. Y.

Also that the following be allowed to retire:

David Choate, of Salem.
Emerson Warner, of Worcester.
Orin Warren, of West Newbury.

Also that the following, having failed to pay their assessments for the past three years, be deprived of the privileges of membership under By-Law VIII.:

Oliver G. Burgess, of Boston.
William J. Chisholm, of Springfield.
Edmond F. Cody, of New Bedford.
Mary P. Eddy, of Beirut, Syria.
Horace S. Everett, of Boston.
Oscar H. Holder, of New York, N. Y.
George W. Huse.
Herbert E. Mace, of Boston.
Timothy E. McOwen, of Lowell.
William E. McPherson, of Charlestown.
Joseph F. Mulcahy, of Lowell.
Benjamin A. Sawyer, of Haverhill.
Ella G. Stone, of Lawrence.
Frederick Tigh, of Newburyport.
Hilbert B. Tingley.
Benjamin N. Towle, of Charlestown.
George F. Webber, of Fairfield, Me.
Sydney Y. Wynne, of Redlands, Cal.

The Committee appointed by the Society in June, 1895, to consider the subject of the condition of instruction in Physiology and Hygiene in the Public Schools of the State and subsequently authorized to report to the Councillors, who should have full power to act thereon, presented a report through their Chairman, Dr. Bowditch, as follows:

The committee sent out a circular of inquiry to the superintendents of schools and the principals of high schools of the State, asking for replies to questions relating to the teaching of physiology and hygiene and of the action of stimulants and narcotics in the schools. Replies were received from 82 cities and towns (scattered over the State), representing 112,000 pupils.

To the question (1) "How are Physiology and Hygiene taught?" the replies showed that in some cases text-books were used, in others the teaching was entirely oral, and in still others there was a combination of the two methods, and that the instruction was ordinarily given by the regular teachers.

To the question (2) "What amount of time is devoted to the teaching of the action of alcohol and narcotics in each school year?" the replies are too indefinite for generalization. In some cases a large part of the time allotted to physiology and hygiene is devoted to a consideration of the effects of stimulants and narcotics, and the study of anatomy, physiology and hygiene becomes incidental. In other cases, the converse is true. The number of the latter, however, is diminished by the fact that in many cases the interpretation and enforcement by local temperance bodies of the law requiring temperance instruction obliges the teacher, contrary to his judgment, to lay the emphasis upon stimulants and narcotics.

The replies to the following questions are summarized in per centages.

(3a) "How is the instruction in the action of alcohol and narcotics given?" Orally, 37%.; text-books, 37%.; experiments, 7%.; combination of these three methods, 17%.; experiments with coagulation of egg albumen by alcohol, 2%.

(3b) "What are the results of this instruction?" Excellent, if well taught, 2%.; good, 11%.; medium, 15%.; little, 14%.; none at all, 55%.; bad, 3%.

(3c) "What value has the instruction in mental and moral discipline?" Great, 1%.; good, if well taught, 16%.; as much as other subjects, 24%.; little, 31%.; none recognized, 28%.

(4) What criticisms of method or matter have occurred to you in your experience in teaching Physiology and Hygiene?" Too much time given to it as required by law, 59%.; statements incorrect and exaggerated, 24%.; more experiments needed, 17%.

(5a) "Have you any change to suggest?" Emphasize moral evil, 8%.; teach less temperance, 14%.; teach more hygiene, 64%.; do not begin physiology before 7th grade, 4%.; do not begin physiology before the high school, 8%.; give special training to teachers, 2%.

These replies were received from superintendents and teachers who showed great interest in the cause of temperance, and who were also in such close contact with the pupils that they were able to judge of the effects of the teaching. The general results of the inquiry may be summed up as follows:

(1) The law requires too much time to be devoted to stimulants and narcotics. The instruction thus becomes, through incessant repetition, perfunctory and meaningless.

(2) Instruction in hygiene, which most teachers regard as more important, is made difficult or impossible through consequent lack of time.

(3) The attempt is made to teach the chemistry and physiology of digestion and nutrition in the grammar and primary schools whose pupils are too young to grasp the necessary conditions.

(4) The knowledge imparted is practically without value because the ideas gained by the pupils are incorrect or exaggerated.

(5) The mental and moral discipline is little or none.

Incidentally it has been shown that the teachers are under such unwelcome pressure on the part of temperance organizations as to be obliged to put what seems to them undue emphasis on the effects of stimulants and narcotics, and consequently inadequate emphasis on the broader aspects of hygienic conditions. Some of the reports came from teachers who, though members of temperance organizations, recognize the danger of the forced temperance instruction. (See appendix A.)

In the course of the investigation by your committee, a hearing was given to prominent teachers in order to get a free expression of their opinions. In this hearing the general feeling seemed to be that there was great pressure upon teachers to teach extreme and often incorrect or exaggerated statements regarding stimulants and narcotics ; and that the ultimate effect of this was to invalidate much of the related teaching. It was emphasized that the systematic teaching of the effects of stimulants and narcotics led, through constant yearly repetition, to a lack of interest on the part of the pupils and to a failure to make moral impressions. It was suggested that formal instruction in physiology and hygiene should not come until the last year of the grammar or in the high school course, and that in primary and grammar grades it was wiser not to use a text-book, but to give occasional pointed talks upon hygienic topics, emphasizing the moral and social dangers of the use of stimulants and narcotics, and encouraging the pupils to avoid danger by abstinence.

A hearing was given to the representatives of the Scientific Temperance Instruction movement, at which they showed a disinclination to have any investigation made of the teaching of physiology and hygiene and of the effects of stimulants and narcotics, and an inclination to consider such investigation antagonistic to themselves. This attitude was shown by the circular letter which they published and sent to all members of The Massachusetts Medical Society and to many others in the State, as also by threatening letters sent to one member of the committee. This misinterpretation of the motives of your committee was persistent and was apparently intended to hamper the investigation to the utmost. If to this policy is added the appeal made through local temperance organizations to the prejudices of people who have little or no knowledge of the methods or principles of teaching, and who are at the same time without the ability to judge properly of the amount and quality of the work which should be given, no further illustration of the methods employed by the advocates of Scientific Temperance Instruction is needed to make clear the pressure brought to bear upon the teachers of physiology and

hygiene in the public schools. The teachers universally sympathize with the motives prompting this interference with their work and so do not make as vigorous resistance to it as they would otherwise. They are further controlled by two threats; first, that they will be judged morally unfit to have the charge of children if they do not voice the extreme views expressed by the temperance organizations, and, second, that pressure will be brought to bear to substitute other teachers who will give these extreme views sympathetic expression.

The effect of this pressure is also shown in the text-books used in the schools. Fifty-seven school systems out of eighty-two heard from use the so-called "Approved Text-Books," that is, text-books in which the amount and character of the so-called temperance instruction have been examined. The "approved text-books" are ordinarily in sets of three: (1) for the primary, (2) for the grammar, and (3) for the high school. They have been submitted by the publishers or authors to a self-constituted committee on Scientific Temperance Instruction. This committee has submitted them in turn to their committee of medical experts, consisting of T. D. Crothers, M.D., Superintendent of an inebriate asylum, and editor of the Journal of Inebriety, Hartford, Conn.; I. M. Quimby, M.D., President of the American Medical Temperance Association and of the Medical Staff of the Jersey City Hospital, Jersey City, N. J.; and L. D. Mason, M.D., Brooklyn, N. Y.

In a circular issued by the temperance organization, it was stated that the text-books were also submitted to Drs. N. S. Davis, Chicago; L. Connor, Detroit; H. O. Marcy, Boston; E. E. Montgomery, Philadelphia; H. D. Holton, Brattleboro; and G. F. Shrady, New York City. Letters were written by your committee to these gentlemen, asking them whether they had examined the books carefully to determine (1) the accuracy of their statements concerning the effects of stimulants and narcotics, (2) the accuracy of the general physiology and hygiene which they contain, and (3) the adaptability of the books to the grades for which they were intended. The replies to these letters indicated that only the accuracy of the statements regarding stimulants and narcotics had been considered at all, and most of them disclaimed agreement with the statements of the books on any ground except that of general accuracy and probable moral benefit.

So far as it is possible to learn, the authors, to receive endorsement by the Scientific Temperance Committee, must devote about one-fifth of the contents of the books to discussions of the nature of stimulants and narcotics and their effects upon the tissues and organs of the body. For the high school physiology the requirement is that at least 20 pages shall be devoted to this instruction. It is not allowable to put this information in an ap-

pendix; the book must specifically state the effects upon each tissue and organ in those portions in which the tissues and organs are described. A pernicious result has been inevitable. Many text-books are written with the sole idea of gaining endorsement through the strength of their total abstinence statements. An examination of these books reveals the fact that there are many definite and positive statements regarding the effects of alcohol which are not recognized by pathologists and have no scientific basis. Many of the statements are not probable or even possible. (See Appendix B.)

The committee desires to call the attention of The Massachusetts Medical Society to an investigation which was undertaken by George H. Martin, one of the agents of the State Board of Education, published in the 55th Annual Report, 1890–91, pp. 309–326. He examined schools in which the instruction had been carried on, and received both oral and written replies. These he classified, and his conclusions are as follows:

(1) The phrase, "scientific temperance instruction," sometimes applied to this work, is a misnomer. There is, and in the nature of things can be, no such instruction. The two essential elements of scientific study — observation and inference — are necessarily wanting; neither the pupil nor the teacher can have first-hand information;

(2) That the outcome in accurate knowledge, resulting from much of the work done, is meagre and out of proportion to the time spent upon it;

(3) That many false impressions are left in the minds of the students;

(4) That physiological details are not suited to young children;

(5) That, however defective the instruction may be, the sentiment of the schools is sound,—the conviction that alcohol and tobacco are bad things to use seems universal;

(6) That the strength of this sentiment does not depend upon the amount of information acquired;

(7) That, where exaggerated notions of the effects of stimulants have been acquired, there is danger of a reaction of sentiment in the light of after-knowledge."

Mr. Martin suggests:

(1) That committees and superintendents give more careful attention to work in this department, prescribing definitely its limits, and requiring the prescribed work to be done as well as work in other subjects, using the same means for judging of its progress and results;

(2) That teachers who are called upon to give oral instruction prepare themselves with great care for the exercise, and see that their statements are true, and by frequent tests, oral and written, ascertain that their teaching is intelligently comprehended by all the pupils;

(3) That, when no text-book is used in any grade, the teachers prepare, for the highest classes, a summary of the effects of stimu-

lants and narcotics upon the different systems of the body, aiming at clearness of statement, and avoiding exaggeration;

(4) That the use of text-books be limited to the older pupils;

(5) That so much of explanation accompany the use of the book as may be necessary to guard against error, and insure exact knowledge;

(6) That, as far as possible, technicalities be avoided;

(7) That the pupils have frequent opportunities to express their knowledge orally and in writing;

(8) That throughout the course the moral and social effects of the use of intoxicants be made prominent, and abstinence be inculcated from higher ends than such as concern only the body.

Your committee heartily endorses these conclusions and suggestions.

The law of Massachusetts regarding temperance education is as follows: Chapter 332, 1885 —

SECTION 1. Physiology and Hygiene, which, in both divisions of the subject, shall include special instruction as to the effect of alcoholic drinks, stimulants and narcotics on the human system, shall be taught as a regular branch of study to all pupils in all schools supported wholly or in part by public money, except special schools maintained solely for instruction in particular branches, such as drawing, mechanics, art and like studies. All acts or parts of acts relating to the qualification of teachers in the public schools shall apply to the branch of study prescribed in this act.

SEC. 2. All penalties now fixed for neglect to provide instruction in the branches of study now prescribed by law shall apply to the branch of study prescribed in Section 1.

SEC. 3. This act shall take effect on the first day of August, in the year eighteen hundred and eighty-five.

One of its authors and its chief advocate in the Senate, thus interprets it:

The object of this Statute is to provide for and secure Physiological Temperance Instruction in the public schools of this Commonwealth.

Physiology and hygiene is a mere method of accomplishing this result. The physiological "effects of alcoholic drinks, stimulants and narcotics" is the predominant and "special" instruction here demanded.

2d. The law further requires that "in both divisions of the subject" of "Physiology and Hygiene," "the effects of alcoholic drinks, stimulants and narcotics" shall be taught. The meaning here is that as the pupil is taught Physiology, or about the structure and the organs of the human body, in the same connection he must be "specially" taught "the effects of alcoholic drinks, stimulants and narcotics" on those organs; and as he studies "Hygiene" or the laws of health, he must in the same connection learn "especially" the relation of alcoholic drinks, stimulants and narcotics to those laws.

3d. The law further specifies that this shall be taught as "a regular branch of the study." "A regular branch" under our

school system is clearly a study which the law requires to be taught, and therefore is regularly found in the public schools. This subject, then, must be taught as other like mandatory studies are taught. If, in any public school, Arithmetic, Grammar, Geography and History (like mandatory studies) are taught with text-books in the hands of pupils, this study, in such a school, must be taught with text-books in like manner. But if the said studies are taught orally in any school, this study in such schools may be taught in the same way.

4th. Again, this branch is not only required in "all public schools" but the law requires it to be taught to "all pupils in all schools" of all grades, and must in all cases be taught as other regular studies are there taught.

For example: In a school where Geography, Grammar, History and Arithmetic are taught with text-books in the hands of the pupils, it would not be compliance with this law to teach this study orally.

Text-books on Physiology and Hygiene which give limited, not "special instruction" as to the "effects of alcoholic drinks, stimulants and narcotics upon the human system," or which put that instruction in an appendix at the end of the book, do not make such an arrangement of the subject as is required by this Statute, and are not such books as school committees are justified in prescribing or teachers in using.

In view of this law; of its extreme interpretation by temperance advocates; of the resulting pressure brought to bear upon authors and publishers of text-books to exaggerate and to distort, upon teachers to devote undue time to the evils of intemperance and insufficient time to the positive aspects of hygiene instruction; of the deadening intellectual influence upon younger pupils of instruction beyond their grasp, upon older pupils of constant uninteresting repetition; and of the well-demonstrated failure to accomplish the moral end universally desired, your committee desires to enter its protest and to recommend a modification of the law to permit rational instruction in anatomy, physiology and hygiene, in the public schools of the State. It further recommends the formulation of a bill for the establishment of a medical commission to pass upon the text-books of anatomy, physiology and hygiene to be used in the public schools of Massachusetts.

H. P. Bowditch.
H. P. Walcott.
G. W. Fitz.

## APPENDIX A.

Quoted from a reply:

I find it impossible to state just how many hours and minutes we give to the subject in our schools, because we keep the *spirit* of the law and *not* the *letter*.

We take the winter term for this instruction and give it sometimes in connection with a reading lesson and sometimes in connection with a language lesson so as to save time.

Both the lady member of the school board, who is its Chairman, and I are strong W. C. T. U. women and wish everything done to emphasize the subject. Yet we honestly think the law is faulty and that the matter is injured in the house of its friends simply through their ignorance of the working of the law.

This reporter gets good results from her work.

## APPENDIX B.—Sample Statements.

The first glass of beer has the power to create a desire for another, until one glass is not sufficient; later on, beer does not satisfy and the stronger drinks are craved, etc.

Primer of Health, Stowell, 1894, p. 74.

Strong drink causes too much blood to flow in their (lungs,) small blood vessels.

Ibid, p. 56.

Because the heart is a muscle it, too, may become fatty from drinking beer. Physicians call this disease "the fatty heart," etc.

Ibid, p. 48.

His (beer drinker's) muscles are changing to fat.

Ibid, p. 33.

Three-fourths of the cases of kidney disease are due to the use of alcohol alone.

Blaisdell's Young Folks Physiology, 1893, p. 241.

* * * The solid part (human body of 75 lbs., dried) would be only about as large as a hen's egg.

The Child's Book of Health, Blaisdell, 1891, p. 38.

When alcohol touches the air cells it thickens the delicate skin and so makes it hard for the air to pass through into the blood. Besides, you can see that the waste matter from the blood finds it just as hard to pass out.

Brand's Good Health for Children, 1890, p. 78.

In accordance with the recommendation of the Committee it was

*Voted*, That in the opinion of the Councillors of The Massachusetts Medical Society, Chapter 332 of the Acts of 1895 should be so amended as to limit instruction in the physiological action of alcohol and narcotics to pupils of the high school grade, and the last year of the grammar school grade.

That a commission should be formed, upon which the medical profession shall be properly represented, to pass upon the text-books on anatomy, physiology and hygiene to be used in the public schools of the State, with the further provision that no text-books which fail to pass this commission shall be used in the schools of the State supported in whole or in part by public

moneys, and that the Committee on State and National Legislation be instructed to support any legislation which will tend to accomplish the purposes above set forth.

*Voted*, That hereafter, until otherwise ordered, the meetings of the Sections be held at 2 P.M., of the Councillors at 5 P.M., and that the Shattuck lecture be delivered at 8 P.M, on the first day of the annual sessions of the Society.

Adjourned at 11.45 A.M.

FRANCIS W. GOSS,
*Recording Secretary.*

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JUNE 13, 1899.

THE ANNUAL MEETING of the Councillors was held in Paul Revere Hall, Mechanic Building, Boston, on Tuesday, June 13, 1899, at 5 o'clock, P.M.

The President, Dr. EDWIN B. HARVEY, in the chair.

The following Councillors were present:

*Barnstable.*
H. S. Kelley, Jr.

*Berkshire.*
J. F. A. Adams,
O. J. Brown,
H. Bushnell.

*Bristol North.*
F. A. Hubbard,
S. D. Presbrey.

*Essex North.*
C. G. Carleton,
I. J. Clarke,
J. A. Douglass,
J. F. Young.

*Essex South.*
F. W. Baldwin,
A. F. Hunt,
W. B. Little,
C. A. Lovejoy,
P. Mooney.

*Hampden.*
E. B. Adams,
D. E. Keefe,
F. F. Parker.

*Middlesex East.*
F. W. Graves,
J. M. Harlow,
J. W. Heath.

*Middlesex North.*
J. A. Gage,
C. B. Sanders,
C. E. Simpson,
W. J. Sleeper.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
E. H. Bigelow,
R. E. Brown,
J. E. Cleaves,
C. H. Cook,
E. Cowles,
J. G. Dearborn,
T. M. Durell,
A. G. Griffin,
E. G. Hoitt,
E. D. Hooker,
R. P. Loring,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
G. A. Oviatt,
C. E. Prior,
L. R. Stone,
A. C. Webber,
J. W. Willis.

*Norfolk.*
H. D. Arnold,

F. A. Bragg,
J. S. Brownrigg,
J. E. Butler,
S. Crowell,
F. C. Dunbar,
D. G. Eldridge,
F. P. Emerson,
I. S. Fogg,
D. H. Fuller,
I. H. Hazelton,
G. H. Ingalls,
G. W. Kaan,
E. B. Lane,
E. G. Morse,
J. J. Sewall.

*Norfolk South.*
W. A. Drake,
J. C. Fraser.

*Plymouth.*
H. W. Dudley,
A. E. Paine.

*Suffolk.*
J. B. Ayer,
E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
B. F. Campbell,
D. W. Cheever,
A. Coolidge. Jr.,
E. G. Cutler,
F. W. Draper,
T. W. Fisher,
C. F. Folsom,
M. F. Gavin,
G. W. Gay,
C. M. Green,
W. H. H. Hastings,
J. Homans,
F. I. Knight,
J. H. McCollom,
A. B. Morong,
A. H. Nichols,
C. B. Porter,
A. Post,
J. J. Putnam,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
G. H. M. Rowe,
G. B. Shattuck,
J. C. White,
E. N. Whittier.

*Worcester.*
W. B. Bowers,
E. B. Harvey,
S. P. Holbrook,
E. W. Norwood,
H. M. Quinby,
E. V. Scribner,
E. R. Wheeler,
L. Wheeler,
A. Wood.

*Worcester North.*
L. G. Chandler,
F. H. Thompson.

Total, 107

The record of the previous meeting was read and accepted.

The Secretary read the names of new and of deceased Fellows.

The Treasurer, Dr. Buckingham, read his annual report.

The Committee on Membership and Finance reported through Dr. Draper and recommended that $4,000.00 of the surplus in the treasury be distributed among the District Societies.

*Voted,* That the recommendation be adopted.

In accordance with the recommendation of the same Committee it was voted, that the following be allowed to resign:

Dwight E. Cone, of Binghamton, N. Y.
Fred B. Jewett, of Howard, R. I.
Nathaniel H. Kirby, of Denver, Col.
John P. Moore, of Long Island City, N. Y.

Also that the following be allowed to retire:

William G. Allen, of Mansfield.
Frederick H. Hooper, of New Bedford.
Edwin A. Kemp, of Danvers.
Samuel J. McDougall, of Boston.
John H. Richardson, of Medfield.
Alexander D. Sinclair, of Boston.

Also that the following be deprived of the privileges of membership under By-Law VIII.:

John L. Ryan, of Malden.

The Committee on Publications presented their report.

The Committee on State and National Legislation reported that while the Committee did not obtain the passage of such an act as was recommended by the Councillors at their last meeting, they did succeed in defeating a bill which was introduced to the Legislature designing to make compulsory, under penalty, the teaching of the physiological action of alcohol and narcotics to the pupils of all grades in the public schools.

The following was presented by Dr. E. G. Cutler:

The Committee appointed to consider the position which schools should maintain in reference to contagious and infectious diseases beg leave to offer the following report, which they advise the Councillors to adopt provisionally for a period of two or three years, and at the end of that time to accept such amendments as seem necessary.

Following a last exposure, provided no illness has resulted, one may return to school after a quarantine lasting as follows: In yellow fever, 5 days; influenza (grippe), 5; diphtheria, 12; measles, 16; small-pox, 18; typhoid fever, 18; typhus fever, 18; chicken-pox, 19; scarlet fever, 20; German measles, 21; whooping-cough, 21; mumps, 25.

When a pupil has had scarlet fever, he may return to school seven weeks from the appearance of the eruption, if desquamation has ceased, the nose and throat are quite healthy and all complications are over, and disinfection of house, patient and belongings has been thoroughly done.

After measles, in three weeks from the appearance of the rash, if desquamation has ceased, and there is no cough, discharge from

the ears, or inflammation of the eyes, and the house, clothes and belongings of the patient have been disinfected.

After mumps, at the end of twenty days, if all glandular swelling has disappeared, and there is no tenderness of the breasts or other part of the body.

After whooping-cough, when all spasmodic cough or whooping has ceased, or whenever cough has entirely left.

After German measles, in two weeks after the commencement of the disease.

After small-pox, when all the scabs are off, especially of the hands and feet, everything that has come in contact with the patient has been burned, and there has been thorough disinfection of everything else.

After chicken-pox, when all the scabs are off, especial care being taken to see that there are no scabs on the scalp, for which purpose the hair should be kept very short and the head thoroughly washed frequently.

After diphtheria, when two consecutive negative cultures from the nose and from the throat have been obtained. When no cultures are taken, a period of 18 or 20 days should elapse after the mucous membrane of the throat has assumed a normal appearance, provided there is no discharge from the nose, before the patient is released from isolation.

After yellow fever, influenza, typhoid fever and typhus fever, at the discretion of the attending physician, who must certify in writing that all danger of contagion is over and proper disinfection of everything has been done.

ELBRIDGE G. CUTLER,
J. H. MCCOLLOM,
J. F. A. ADAMS,
} *Committee.*

*Voted*, That the report be accepted and printed in the "Proceedings."

The Librarian, Dr. Brigham, presented his annual report.

The Committee on Nominations reported a list of candidates for the offices of the Society for the ensuing year, and the same were elected by ballot:

| | |
|---|---|
| *President* . . | Dr. EDWIN B. HARVEY, of Westboro'. |
| *Vice-President* . | Dr. CHARLES M. GREEN, of Boston. |
| *Treasurer* . . | Dr. EDWARD M. BUCKINGHAM, of Boston. |
| *Corresponding Sec'y* | Dr. CHARLES W. SWAN, of Brookline. |
| *Recording Secretary* | Dr. FRANCIS W. GOSS, of Roxbury. |
| *Librarian* . . | Dr. EDWIN H. BRIGHAM, of Brookline. |

Dr. ARTHUR T. CABOT, of Boston, was chosen Orator for the annual meeting of the Society in 1900.

*Voted*, That the next annual meeting of the Society be held in Boston on the second Wednesday in June, 1900.

The following Standing Committees were appointed:

*Of Arrangements.*

J. E. Goldthwait, S. Crowell, F. G. Balch,
M. Storer, W. H. Prescott, J. C. Hubbard.

*On Publications.*

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

*On Membership and Finance.*

F. W. Draper, E. G. Cutler, L. R. Stone,
F. W. Goss, W. Ela.

*To Procure Scientific Papers.*

H. L. Burrell, A. K. Stone, F. H. Thompson,
G. de N. Hough, H. W. Van Allen.

*On Ethics and Discipline.*

G. E. Francis, F. C. Shattuck, C. G. Carleton,
E. Cowles, J. F. A. Adams.

*On Medical Diplomas.*

H. E. Marion, E. N. Whittier, O. F. Rogers.

*On State and National Legislation.*

E. B. Harvey, H. P. Bowditch, T. H. Gage,
S. D. Presbrey, S. W. Abbott.

Adjourned at 6.05 P.M.

FRANCIS W. GOSS,
*Recording Secretary.*

# The Massachusetts Medical Society.

## PROCEEDINGS OF THE SOCIETY.

### ANNUAL MEETING.

### First Day.

### June 13, 1899.

Sectional meetings were held in Mechanic Building, Boston, on Tuesday, June 13, 1899, at 2 o'clock, P.M.

The sections were organized and papers were presented as follows:

#### SECTION IN MEDICINE.

Dr. C. F. Folsom, of Boston . . . . . Chairman.
Dr. A. K. Stone, of Boston . . . . . Secretary.

The Clinical Importance of a Knowledge of the Size of the Heart.—By Dr. F. H. Williams, of Boston.

The Diagnostic and Prognostic Value of the Pulse.—By Dr. Henry Jackson, of Boston.

Preliminary Note on the Prognosis of Nephritis.—By Drs. R. C. Cabot and F. W. White, of Boston.

The Rôle of the Staphylococcus in Skin Diseases.—By Dr. C. J. White, of Boston.

#### SECTION IN SURGERY.

Dr. E. H. Bradford, of Boston . . . . Chairman.
Dr. F. G. Balch, of Boston . . . . . Secretary.

Intussusception.—By Dr. J. C. Munro, of Boston.

THE COMPLETED HISTORY OF A CASE OF GASTRECTOMY. REMARKS UPON THE SURGERY OF GASTRIC CANCER.—By Dr. M. H. Richardson, of Boston.

A LANTERN-SLIDE DEMONSTRATION OF THE DISSEMINATION OF CARCINOMA.—By Dr. W. T. Councilman, of Boston.

At 8 o'clock, P.M., the Shattuck Lecture was delivered by Dr. JAMES J. PUTNAM, of Boston.

Following the lecture a reception was given by the Society to its officers.

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## SECOND DAY.

### JUNE 14, 1899.

THE Society met in Mechanic Building, Boston, on Wednesday, June 14, 1899, at 9 o'clock, A.M., for the exercises of the one hundred and eighteenth Anniversary.

The President, Dr. EDWIN B. HARVEY, in the chair.

The record of the last annual meeting was read and accepted.

The Secretary read the names of Fellows reported admitted since the last annual meeting, and of those whose deaths had been recorded.

*Admissions reported since June 7, 1898.*

| | | |
|---|---|---|
| 1898 | Adams, John | Brookline. |
| 1899 | Adams, William Carlton | East Taunton. |
| 1899 | Allen, Clarence Jean | Winchester. |
| 1898 | Allen, Seabury Wells | Boston. |
| 1898 | Anthony, Jeremiah Christopher | Springfield. |
| 1899 | Bacon, Theodore Spaulding | Natick. |
| 1899 | Badger, George Sherwin Clarke | Brookline. |
| 1898 | Bailey, Walter Channing | Somerville. |
| 1898 | Baker, Benjamin Ward | Taunton. |
| 1899 | Barrell, Mary Elizabeth | Worcester. |
| 1899 | Bartlett, Charles Watson | Marshfield. |
| 1899 | Bartlett, Samuel Danforth | Woburn. |
| 1898 | Beattie, Robert Fowler | Swampscott. |

| | | |
|---|---|---|
| 1898 | Benson, Charles Sweetser | Haverhill. |
| 1898 | Berg, Telka Amalia Josefina | Lynn. |
| 1898 | Blodgett, John Hammond | Boston. |
| 1898 | Brickett, Beatrice Hannah | Newton Centre. |
| 1898 | Briggs, Charles Edwin | Cleveland, Ohio. |
| 1898 | Broughton, Arthur Nicholson | Jamaica Plain. |
| 1899 | Brown, Daniel Rollins | Salem. |
| 1898 | Brown, Edward Wells | Springfield. |
| 1899 | Buehler, George Van Buskirk | Boston. |
| 1898 | Buffum, Herbert Edwin | Boston. |
| 1899 | Butler, Charles Shorey | Boston. |
| 1898 | Cabot, Hugh | Boston. |
| 1899 | Chamberlin, Theodore | Concord. |
| 1899 | Churchill, Donald | Jamaica Plain. |
| 1898 | Clark, Albert Ulysses Franklin | Wilmington. |
| 1898 | Coghlan, John Nicholas | Holyoke. |
| 1899 | Collins, William James | Northampton. |
| 1898 | Coon, George Bailey | Taunton. |
| 1899 | Crandon, Le Roi Goddard | Boston. |
| 1899 | Cronin, Michael John | Boston. |
| 1898 | Cronin, Thomas Joseph | Webster. |
| 1899 | Cross, William Patrick | South Boston. |
| 1898 | Curran, Charles Henry | Holyoke. |
| 1899 | Curtis, William Goodwin | Wollaston. |
| 1898 | Cusick, Thomas Francis | Taunton. |
| 1899 | Cummin, John White | Boston. |
| 1898 | Davis, William Horace | Holyoke. |
| 1899 | Davis, Winifred | Charlestown. |
| 1898 | Dean, Charles Henry | Dorchester. |
| 1898 | Dennett, Daniel Clement | Winchester. |
| 1899 | Donoghue, James Crowley | Boston. |
| 1899 | Dow, David Crooker | Cambridge. |
| 1899 | Downey, William Henry | Peabody. |
| 1899 | Drake, Arthur Knowlton | Tewksbury. |
| 1899 | Ellis, William Raymond | Allston. |
| 1899 | Ellsworth, Samuel Walker | Quincy. |
| 1899 | Emerson, Ernest Benjamin | Tewksbury. |
| 1899 | Everett, Eugene Ellsworth | Boston. |
| 1899 | Fair, John Francis | Boston. |
| 1899 | Fairbanks, Arthur Willard | Boston. |
| 1898 | Fernald, Alberto Francis | Shirley. |
| 1899 | Field, Harvey Adams | Dorchester |
| 1898 | Fitzgerald, Clara Pauline | Worcester. |
| 1899 | Forrest, Robert Francis | Cambridge. |
| 1898 | Frost, Flora Eva | Granby. |
| 1898 | Frost, Samuel Knapp | Pittsfield. |
| 1898 | Fullerton, Walter Wilson | Brockton. |

| | | |
|---|---|---|
| 1899 | Gallagher, William Howard | Malden. |
| 1899 | Garland, George Franklin | Waltham. |
| 1899 | Gavin, John Harrison | Roxbury. |
| 1898 | Gibbons, Sherwin | Lexington. |
| 1899 | Grady, Patrick Anthony Salmon | Clinton. |
| 1898 | Greenough, Robert Battey | Boston. |
| 1899 | Grimes, James Henry | Boston. |
| 1898 | Halloran, Timothy Joseph | Lowell. |
| 1899 | Hamilton, Annie Lee | Boston. |
| 1898 | Harvey, William Wirt | Roxbury. |
| 1899 | Haviland, Nathaniel Clark Bacon | Holliston. |
| 1898 | Hayes, Albert Edwin | Lowell. |
| 1899 | Hayes, Frederick Legro | Roxbury. |
| 1899 | Healy, Thomas Raymond | Newburyport. |
| 1899 | Hinchey, Richard | Roxbury. |
| 1899 | Hinckley, James William | Boston. |
| 1898 | Holland, William Timothy | Winchester. |
| 1898 | Howard, Eugene Henry | Pittsfield. |
| 1899 | Howe, Joseph Dimock | Becket. |
| 1898 | Howe, Walter Clarke | Dedham. |
| 1898 | Hoyt, Dixi George | Providence, R. I. |
| 1898 | Huntington, Alfred Thomas | Boston. |
| 1899 | Hyde, Frederick Tanquary | Waltham. |
| 1899 | Jackson, Alexander Washington | Dorchester. |
| 1898 | Janes, George Herbert | Westfield. |
| 1899 | Jenness, Burt Franklin | Tewksbury. |
| 1898 | Johnson, Herbert Shattuck | Malden. |
| 1898 | Judkins, Anna Gertrude | Roxbury. |
| 1898 | Kennedy, Alexander Gladstone | Boston. |
| 1898 | Kimball, James Henry | Greenwood. |
| 1898 | King, Myron Louis | Cambridge. |
| 1899 | Knight, Howard Webster | Dorchester. |
| 1899 | Lecompte, Walter Augustus | Newton Centre. |
| 1898 | Leonard, Edwin, Jr. | Melrose Highlands. |
| 1899 | Libby, Mary Gordon | Lowell. |
| 1898 | Lockary, Joseph Logue | Roxbury. |
| 1898 | Lockwood, Charles Edwin | Lynn. |
| 1898 | Lowell, Alverne Percy | Fitchburg. |
| 1899 | McAllister, Frederick Danforth | Worcester. |
| 1899 | Macdonald, Donald William | Brookline. |
| 1899 | Mackie, William Charles | Worcester. |
| 1898 | Mackintosh, Ernest Robert | Lowell. |
| 1898 | McLaughlin, James Stephen | Westfield. |
| 1898 | McNeish, Alexander | Leicester. |
| 1898 | McQuaid, Thomas Bernard | Everett. |
| 1898 | Mahoney, Edward Joseph | Holyoke. |
| 1899 | Mansur, Leon Wallace | Boston. |

| | | |
|---|---|---|
| 1898 | Marston, Joseph Norris | Lowell. |
| 1898 | Marvin, Sydney Robertson | Springfield. |
| 1899 | May, John Shepard | Roxbury. |
| 1898 | May, William Ropes | Boston. |
| 1898 | Meek, Edith Ruth Eliza | Boston. |
| 1899 | Merrill, Quincy Heald | Fitchburg. |
| 1898 | Moore, John Henry | Boston. |
| 1898 | Morse, Almon Gardner | Boston. |
| 1898 | Mosher, Harris Peyton | Boston. |
| 1898 | Musgrave, Percy | Boston. |
| 1898 | O'Connor, John James | Holyoke. |
| 1899 | O'Keefe, Daniel Thomas | Jamaica Plain. |
| 1899 | O'Sullivan, John Joseph | Lawrence. |
| 1899 | Overlock, Melvin George | Worcester. |
| 1899 | Packard, George Henry | Medford. |
| 1899 | Page, Frederick Carroll | Brookline. |
| 1899 | Parker, Ralph Walker | Lowell. |
| 1899 | Parker, Walter Henry | Dorchester. |
| 1899 | Pease, Charles Wood | Tewksbury. |
| 1898 | Peirce, Frederic Joseph | Quincy. |
| 1899 | Peirce, George Alphonso | Tewksbury. |
| 1899 | Pettee, John Harris | Roxbury. |
| 1898 | Pierce, George Burgess | Milton. |
| 1898 | Pigeon, Walter Hugh | Lynn. |
| 1898 | Plummer, Frank Wentworth | Malden. |
| 1898 | Pool, Charles Bret | Lowell. |
| 1899 | Potter, Lester Forest | New Bedford. |
| 1898 | Potts, Joseph Henry | Holyoke. |
| 1898 | Pratt, Joseph Hersey | Boston. |
| 1898 | Proctor, Joseph Whipple | Malden. |
| 1899 | Reagh, Arthur Lincoln | West Roxbury. |
| 1898 | Reed, Victor Augustus | Lawrence. |
| 1899 | Reid, Eustace Palmer | Boston. |
| 1898 | Richmond, Simon | Boston. |
| 1898 | Ring, Arthur Hallam | Arlington Heights. |
| 1898 | Robie, Walter Franklin | Baldwinsville. |
| 1898 | Ross, Ellsworth Frank | Wales. |
| 1898 | Russell, Edward Ervin | North Adams. |
| 1898 | Schneider, Jacob Philip | Palmer. |
| 1899 | Shurtleff, Walter Davis | Carver. |
| 1898 | Smith, Alfred Charles | Everett. |
| 1898 | Smith, William Henry | Boston. |
| 1898 | Stetson, Frank Eliot | New Bedford. |
| 1898 | Stodder, Charles William | Marshfield Hills. |
| 1899 | Strong, Frederick Finch | Boston. |
| 1898 | Sturgis, Benjamin Franklin, Jr. | Salem. |
| 1899 | Sughrue, Dennis Francis | Roxbury. |

| | | |
|---|---|---|
| 1899 | Tenney, Elmer Seth | Roxbury. |
| 1898 | Thompson, Peter Hunter | Boston. |
| 1899 | Thompson, Richard Henry | Malden. |
| 1899 | Thorn, Edwin Cyrus | Deerfield. |
| 1899 | Walker, Alexander Taylor | Falmouth. |
| 1898 | Wallace, George Loney | Waverley. |
| 1899 | Walsh, Charles Joseph | Cambridge. |
| 1898 | Ward, Parker Myles | Dorchester. |
| 1899 | Watson, Frank Gilman | Roxbury. |
| 1898 | Weis, Joseph Deutsch | Boston. |
| 1898 | Wetherell, Arthur Bryant | Holyoke. |
| 1899 | White, Michael William | Boston. |
| 1898 | Wilson, Robert Browning | Hubbardston. |
| 1899 | Wood, Mary Anna | Pittsfield. |
| 1899 | Woodbury, Frank Taylor | Wakefield. |
| 1899 | Young, Emma Elizabeth | Chelsea. |

Total, 170.

## *List of Deceased Fellows.*

| Admitted. | Name. | Residence. | Date of Death. | Age. |
|---|---|---|---|---|
| 1889 | *Anagnostakis, Andreas | Athens, Greece. | | |
| 1892 | Barss, James Richmond | Malden | Oct. 24, 1898 | 48 |
| 1871 | Belt, Charles Bradford | South Boston | Aug. 23, 1898 | 50 |
| 1866 | Bigelow, Orvis Furman | Amherst | Feb. 2, 1899 | 63 |
| 1866 | Blood, Josiah Mark | Ashby | Nov. 19, 1898 | 66 |
| 1894 | Bowen, Henry Charles | Springfield | Aug. 13, 1898 | 30 |
| 1875 | Bowen, Seranus | Roxbury | April 7, 1899 | 59 |
| 1867 | Bridgman, Marcus Fayette | Boston | Jan. 20, 1899 | 75 |
| 1866 | Brigham, Frank Whiting | Shrewsbury | Feb. 28, 1899 | 57 |
| 1839 | Browne, Charles Hale | Boston | Feb. 28, 1898 | 85 |
| 1874 | Clement, Thomas Runnells | Osterville | Sept. 18, 1898 | 75 |
| 1869 | Clough, Benjamin Franklin | Worcester | May 8, 1899 | 61 |
| 1874 | Coburn, George Albert. | East Cambridge. | Sept. 2, 1898 | 52 |
| 1854 | Colony, George Danforth | Fitchburg | Oct. 2, 1898 | 77 |
| 1892 | Curtis, John Benedict | East Somerville. | Oct. 1, 1898 | 31 |
| 1896 | Dewey, David Robert | North Adams | Nov. 5, 1898 | 34 |
| 1856 | Fobes, Joseph Bassett | Bridgewater | Aug. 30, 1898 | 83 |
| 1895 | Fox, Charles Leonard | Lowell | Oct. 5, 1898 | 28 |
| 1875 | Freeman, George Edward. | Brockton | June 12, 1898 | 56 |
| 1873 | Frost, George Melville | Peabody | June 20, 1898 | 56 |
| 1866 | Gamwell, Harlow | Westfield | Aug. 11, 1898 | 63 |
| 1858 | Godding, William Whitney | Washington, D.C | May 6, 1899 | 68 |
| 1890 | Holmes, Charles Moses | Northampton | Oct. 2, 1898 | 36 |
| 1898 | Huntington, Alfred Thomas | Boston | Feb. 13, 1899 | 31 |
| 1870 | Hurd, Edward Payson | Newburyport | Feb. 24, 1899 | 60 |
| 1872 | Jackson, Eben | Somerville | June 22, 1898 | 73 |
| 1863 | Kemble, Arthur | Salem | Oct. 27, 1898 | 59 |
| 1893 | Kidder, Edward Hamilton | Fall River | July 16, 1898 | 33 |
| 1851 | Knowlton, Charles Lorenzo | Northampton | July 5, 1898 | 74 |
| 1897 | Lawrence, James Christy Hurd. | Boston | Sept. 22, 1897 | 32 |

*Honorary.

| | | | | | |
|---|---|---|---|---|---|
| 1856 | Lovejoy, Oliver Sherwin | Haverhill | June | 26, 1898 | 72 |
| 1885 | Lynch, Samuel Bartlett | Boston | Jan. | 14, 1899 | 38 |
| 1894 | McMillan, Wallace | Dorchester | March | 31, 1899 | 31 |
| 1896 | Manson, Frank George | Billerica | Oct. | 19, 1898 | 32 |
| 1845 | Minot, Francis | Boston | May | 11, 1899 | 78 |
| 1894 | Moore, William Morrison | Provincetown | June | 10, 1898 | 49 |
| 1889 | O'Connor, Thomas Henry | Clinton | Jan. | 3, 1899 | 42 |
| 1893 | Pick, Albert Edward | Brockton | Feb. | 12, 1899 | 34 |
| 1867 | Quincy, Henry Parker | Dedham | March | 11, 1899 | 60 |
| 1866 | Rockwell, Joseph William | Southwick | Dec. | 31, 1893 | 83 |
| 1897 | Ronayne, David Patrick | Boston | April | 2, 1899 | 32 |
| 1848 | Sparrow, William Edward | Mattapoisett | May | 15, 1899 | 75 |
| 1869 | Stone, William Newcomb | Wellfleet | Oct. | 17, 1898 | 53 |
| 1867 | Vermyne, Jan Joseph Bastianus | New Bedford | Aug. | 16, 1898 | 63 |
| 1872 | Vogel, Frederick William | Roxbury | July | 16, 1898 | 52 |
| 1873 | Walsh, Edmund | East Cambridge | Oct. | 19, 1898 | 50 |
| 1888 | Worcester, Charles Pomeroy | Newtonville | Oct. | 9, 1898 | 37 |

Total, 47.

The Treasurer's report was presented.

Papers were read as follows:

Improvements in Army Medical Service, suggested by Experience in the Late War.—By Dr. W. H. Devine, Lieutenant Colonel and Medical Director, Second Brigade, M. V. M., late Major and Brigade Surgeon, U. S. V.

A Report of the Blood Examinations in Typhoid Fever and in the Malarial Fevers.—By Dr. J. J. Curry, Acting Assistant Surgeon, U. S. A., Pathologist to the U. S. A. General Hospital, Savannah, Ga.

A Hospital Clearing House.—By Dr. C. J. Blake, of Boston.

Demonstration of Parasites in the Blood and Tissues in Malaria.—By Dr. J. H. Wright, of Boston.

Methods of Conducting Volunteer Aid in Foreign Territory.—By Dr. E. G. Brackett, of Boston.

Drs. J. H. McCollom and E. J. Cutler, of Boston, gave a stereopticon exhibit illustrating clinical medicine and small-pox.

At 12 o'clock the Annual Discourse was delivered by Dr. Edward H. Bradford, of Boston.

*Voted*, That the thanks of the Society be presented to the Orator for his able and instructive address.

At 1 p.m. the Annual Dinner was served to more than nine hundred Fellows.

FRANCIS W. GOSS,
*Recording Secretary.*

# TREASURER'S REPORT.

APRIL 15, 1899.

*Receipts.*

| | | | |
|---|---|---|---|
| Balance from previous year | | | $6,914 78 |
| Assessments— | | | |
| Paid at Annual Meeting | | $690 00 | |
| Paid to Treasurer at other times | | 460 00 | |
| Paid to District Treasurers of: | | | |
| Barnstable | $115 00 | | |
| Berkshire | 295 00 | | |
| Bristol North | 175 00 | | |
| Bristol South | 345 00 | | |
| Essex North | 345 00 | | |
| Essex South | 525 00 | | |
| Franklin | 115 00 | | |
| Hampden | 505 00 | | |
| Hampshire | 225 00 | | |
| Middlesex East | 195 00 | | |
| Middlesex North | 460 00 | | |
| Middlesex South | 1,260 00 | | |
| Norfolk | 1,075 00 | | |
| Norfolk South | 200 00 | | |
| Plymouth | 255 00 | | |
| Suffolk | 2,425 00 | | |
| Worcester | 805 00 | | |
| Worcester North | 205 00 | | |
| | $9,525 00 | 9,525 00 | |
| | | $10,675 00 | 10,675 00 |
| Income :— | | | |
| Interest on Bank Accounts— | | | |
| Mount Vernon National Bank | $114 97 | | |
| Bay State Trust Co. | 62 36 | | |
| | $177 33 | 177 33 | |
| Interest on Savings Bank Accounts— | | | |
| Franklin Savings Bank (Cotting Fund)— | $37 92 | | |
| Provident Inst. for Savings | 35 30 | | |
| Roxbury Inst. for Savings | 35 30 | | |
| Suffolk Savings Bank | 32 76 | | |
| | $141 28 | 141 28 | |
| Interest on Bonds (Phillips Fund)— | | | |
| United States Bond | $400 00 | | |
| Massachusetts Bonds | 70 00 | | |
| | $470 00 | 470 00 | |
| *Amounts carried forward,* | | $788 61 | $17,589 78 |

| | | | |
|---|---|---|---|
| *Amounts brought forward,* | | $788 61 | $17,589 78 |
| Interest on Annuities of Mass. Hosp. Life Ins. Co.— | | | |
| Belonging to Permanent Fund | $450 12 | | |
| Belonging to Shattuck Fund | 366 67 | | |
| | $816 79 | 816 79 | |
| | | $1,605 40 | $1,605 40 |
| Sale of Extra Dinner Tickets | | | 8 00 |
| Fee for Engraved Diploma | | | 5 00 |
| | | | $19,208 18 |
| *Expenses.* | | | |
| Twice paid assessments returned | | $5 00 | |
| District Treasurers' Expense | | 615 15 | |
| Treasurer's Expense | | 98 80 | |
| Librarian's Expense | | 113 48 | |
| Custodian's Expense | | 2 00 | |
| Catalogue | | 11 40 | |
| Secretary's Expense | | 313 31 | |
| Censors' Expense | | 486 55 | |
| Supervisors' Expense | | 16 98 | |
| Committee on Publications | | 1,047 47 | |
| Cotting Lunch | | 100 50 | |
| Shattuck Lecture | | 201 25 | |
| Annual Meeting :— | | | |
| Caterer | $1,765 56 | | |
| Cigars | 97 50 | | |
| Rent of Hall | 596 35 | | |
| Police | 9 87 | | |
| Construction of Platform | 75 00 | | |
| Card Signs | 10 15 | | |
| Boston Elevated Railroad | 20 10 | | |
| Music | 40 00 | | |
| Stationery | 51 60 | | |
| Addressing Circulars | 16 00 | | |
| Lantern Service | 11 00 | | |
| Stereopticon Service | 23 50 | | |
| Carriage Hire | 1 00 | | |
| Stenographer | 6 00 | | |
| Incidentals | 20 39 | | |
| | $2,744 02 | 2,744 02 | |
| Salaries | | 1,400 00 | |
| Rent | | 150 00 | |
| Annual Dividend :— | | | |
| Barnstable | $35 19 | | |
| Berkshire | 68 61 | | |
| *Amounts carried forward,* | $103 80 | $7,306 91 | $19,208 18 |

| | | | |
|---|---|---|---|
| *Amounts brought forward,* | $103 80 | $7,306 91 | $19,208 18 |
| Bristol North | 42 23 | | |
| Bristol South | 107 83 | | |
| Essex North | 124 93 | | |
| Essex South | 151 32 | | |
| Franklin | 35 19 | | |
| Hampden | 124 93 | | |
| Hampshire | 79 18 | | |
| Middlesex East | 59 83 | | |
| Middlesex North | 133 73 | | |
| Middlesex South | 429 32 | | |
| Norfolk | 374 78 | | |
| Norfolk South | 61 59 | | |
| Plymouth | 79 18 | | |
| Suffolk | 781 22 | | |
| Worcester | 251 61 | | |
| Worcester North | 59 83 | | |
| | $3,000 00 | 3,000 00 | |
| | | $10,306 91 | 10,306 91 |
| | Balance April 15, 1899 | | $8,901 27 |
| Due to the Cotting Fund | | | 30 76 |
| Leaving for the general purposes of the Society | | | $8,870 51 |

*Permanent Investments.*

The permanent investments of the Society consist of :—

| | |
|---|---|
| Three annuity policies Massachusetts Hospital Life Insurance Co. | $11,253 30 |
| One annuity policy Massachusetts Hospital Life Insurance Co. (Shattuck Fund) | 9,166 87 |
| United States 4 per cent. bond (Phillips Fund) | 10,000 00 |
| Massachusetts 3½ per cent. bonds | 2,000 00 |
| Deposit book, Franklin Savings Bank | 1,074 48 |
| Deposit Book, Institution for Savings in Roxbury (Cotting Fund) | 1,000 00 |
| Deposit book, Provident Institution for Savings (Cotting Fund) | 1,000 00 |
| Deposit book, Suffolk Savings Bank (Cotting Fund) | 1,000 00 |
| Total, | $36,494 65 |

EDWARD M. BUCKINGHAM,
*Treasurer.*

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We, the undersigned, a duly appointed Committee, having examined the books of the Treasurer, find them correctly cast and properly vouched ; and also that he has in his possession the various securities called for.

F. GORDON MORRILL.
H. M. CUTTS.

MAY 29, 1899.

## Officers of The Massachusetts Medical Society.

1899—1900.

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Chosen June 13, 1899.

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EDWIN B. HARVEY, . . . Westboro', President.
CHARLES M. GREEN, . . Boston, . Vice-President.
EDWARD M. BUCKINGHAM, Boston, . Treasurer.
CHARLES W. SWAN, . . . Boston, . Cor. Secretary.
FRANCIS W. GOSS, . . . Roxbury, Rec. Secretary.
EDWIN H. BRIGHAM, . . Boston, . Librarian.

### Standing Committees.

*Of Arrangements.*

J. E. Goldthwait, S. Crowell, F. G. Balch,
M. Storer, W. H. Prescott, J. C. Hubbard.

*On Publications.*

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

*On Membership and Finance.*

F. W. Draper, E. G. Cutler, L. R. Stone,
F. W. Goss, W. Ela.

*To Procure Scientific Papers.*

H. L. Burrell, A. K. Stone, F. H. Thompson,
G. de N. Hough, H. W. Van Allen.

*On Ethics and Discipline.*

G. E. Francis, F. C. Shattuck, C. G. Carleton,
E. Cowles, J. F. A. Adams.

*On Medical Diplomas.*

H. E. Marion, E. N. Whittier, O. F. Rogers.

*On State and National Legislation.*

E. B. Harvey, H. P. Bowditch, T. H. Gage,
S. D. Presbrey, S. W. Abbott.

## Presidents of District Societies—Vice-Presidents (Ex-Officiis).

[Arranged according to seniority.]

| | |
|---|---|
| M. G. Parker, | E. D. Hill, |
| E. H. Stevens, | W. Holbrook, |
| W. Davis, | S. E. Greenwood, |
| G. W. Tinkham, | C. A. Atwood, |
| J. J. Healy, | W. S. Birge, |
| E. P. Gerry, | H. H. Seelye, |
| J. E. Garland, | W. W. Scofield, |
| J. P. Bixby, | A. P. Webber, |
| H. L. Burrell, | M. P. Dole. |

## Councillors.

Barnstable.—Drs. R. H. Faunce, Sandwich; H. S. Kelley, West Dennis.

Berkshire.—Drs. J. F. A. Adams, Pittsfield; O. J. Brown, H. Bushnell, North Adams; W. W. Leavitt, F. K. Paddock, Pittsfield.

Bristol North.—Drs. F. A. Hubbard, S. D. Presbrey, Taunton; A. M. Round, Norton.

Bristol South.—Drs. J. T. Bullard, New Bedford; A. B. Cushman, South Dartmouth; G. L. Ellis, Middleboro'; G. S. Eddy, J. H. Gifford, J. H. Jackson, Fall River; G. de N. Hough, C. D. Prescott, New Bedford.

Essex North.—Drs. G. G. Carleton, Lawrence; I. J. Clarke, C. E. Durant, Haverhill; J. A. Douglass, Amesbury; J. F. Richards, Andover; R. B. Root, Georgetown; O. Warren, West Newbury; J. F. Young, Newburyport.

Essex South.—Drs. F. S. Atwood, Salem; F. W. Baldwin, A. H. Harrington, Danvers; H. K. Foster, Peabody; C. W. Haddock, Beverly; A. F. Hunt, Swampscott; W. B. Little, C. A. Lovejoy, C. E. Meader, Lynn; P. Mooney, Gloucester; E. L. Peirson, S. F. Quimby, Salem.

Franklin.—Drs. F. J. Canedy, Shelburne Falls; W. H. Pierce, A. C. Walker, Greenfield.

Hampden.—Drs. E. B. Adams, E. A. Bates, S. D. Brooks, C. H. Calkins, Springfield; F. W. Ellis, Monson; F. Holyoke,

Holyoke; C. P. Hooker, Springfield; C. W. Jackson, Monson; D. E. Keefe, W. H. Pomeroy, Springfield; F. F. Parker, Chicopee.

HAMPSHIRE.—Drs. N. C. Haskell, Amherst; J. G. Hayes, Williamsburg; C. Seymour, Northampton; J. Stowell, North Amherst.

MIDDLESEX EAST.—Drs. F. W. Graves, J. M. Harlow, Woburn; J. W. Heath, Wakefield; G. W. Nickerson, Stoneham.

MIDDLESEX NORTH.—Drs. W. G. Eaton, J. B. Field, J. A. Gage, L. Huntress, C. B. Sanders, C. E. Simpson, Lowell; W. J. Sleeper, Westford; C. A. Viles, Lowell.

MIDDLESEX SOUTH.—Drs. Z. B. Adams, E. H. Bigelow, Framingham; D. E. Baker, Newtonville; R. E. Brown, Everett; J. E. Cleaves, Medford; C. H. Cook, Natick; E. Cowles, Waverley; R. A. Blood, J. G. Dearborn, Charlestown; T. M. Durell, Somerville; A. G. Griffin, Malden; E. G. Hoitt, Marlboro'; E. D. Hooker, Arlington; R. P. Loring, Newton Centre; H. E. Marion, Brighton; J. T. G. Nichols, A. L. Norris, Cambridge; G. A. Oviatt, South Sudbury; G. C. Pierce, Ashland; C. E. Prior, Malden; E. H. Stevens, Cambridge; L. R. Stone, Newton; H. P. Walcott, A. C. Webber, M. Wyman, Cambridge; J. W. Willis, Waltham.

NORFOLK.—Drs. H. D. Arnold, Roxbury; F. A. Bragg, Foxboro'; J. S. Brownrigg, J. E. Butler, Roxbury; C. A. Cheever, S. Crowell, Dorchester; H. M. Cutts, Brookline; F. C. Dunbar, Roxbury; D. G. Eldridge, Dorchester; F. P. Emerson, Roxbury; I. S. Fogg, Norwood; D. H. Fuller, Jamaica Plain; I. H. Hazelton, Wellesley Hills; H. R. Hitchcock, Hyde Park; S. A. Houghton, Brookline; G. H. Ingalls, Jamaica Plain; G. W. Kaan, Brookline; E. B. Lane, Roslindale; A. Mitchell, Medfield; E. G. Morse, Roxbury; H. V. Reynolds, Dorchester; G. K. Sabine, Brookline; J. J. Sewall, Roxbury; L. F. Vickery, Jamaica Plain.

NORFOLK SOUTH.—Drs. W. A. Drake, North Weymouth; J. C. Fraser, East Weymouth; J. F. Welch, Quincy.

PLYMOUTH.—Drs. H. W. Dudley, Abington; B. F. Hastings, Whitman; N. K. Noyes, Duxbury; A. E. Paine, Brockton; F. G. Wheatley, North Abington.

SUFFOLK.—Drs. J. B. Ayer, E. M. Buckingham, *Treasurer*, H. L. Burrell, J. F. Bush, A. T. Cabot, B. F. Campbell, D. W. Cheever, A. Coolidge, Jr., E. G. Cutler, H. Derby, F. W. Draper, T. W. Fisher, R. H. Fitz, C. F. Folsom, W. W. Gannett, Boston; M. F. Gavin, South Boston; G. W. Gay, C. M. Green, W. H. H. Hastings, J. Homans, B. J. Jeffries, G. F. Jelly, F. I. Knight, A. L. Mason, J. H. McCollom, A. B. Morong, F. G. Morrill, A. H. Nichols, C. B. Porter, A. Post, J. J. Putnam, Boston; J. M. Putnam, Chelsea; M. H. Richardson, W. L. Richardson, T. M. Rotch, G. H. M. Rowe, F. C. Shattuck, G. B. Shattuck, A. M. Sumner, J. B. Swift, G. G. Tarbell, H. F. Vickery, J. C. Warren, J. C. White, E. N. Whittier, Boston.

WORCESTER.—Drs. W. P. Bowers, Clinton; P. P. Comey, W. Davis, G. E. Francis, T. H. Gage, Worcester; E. B. Harvey, *President*, Westboro'; S. P. Holbrook, East Douglas; J. O. Marble, H. M. Quinby, Worcester; W. G. Reed, Southbridge; E. W. Norwood, E. R. Wheeler, Spencer; E. V. Scribner, L. Wheeler, A. Wood, Worcester.

WORCESTER NORTH.—Drs. L. G. Chandler, Townsend; E. J. Cutter, Leominster; B. H. Hartwell, Ayer; F. H. Thompson, Fitchburg.

## Censors.

BARNSTABLE.—Drs. G. W. Doane, Hyannis; R. H. Faunce, *Supervisor*, Sandwich; G. N. Munsell, Harwich; S. Pitcher, Hyannis; F. A. Rogers, Chatham.

BERKSHIRE.—Drs. H. Bushnell, North Adams; H. Colt, W. W. Leavitt, *Supervisor*, W. M. Mercer, Pittsfield; W. W. Scofield, Dalton.

BRISTOL NORTH.—Drs. C. A. Atwood, F. A. Hubbard, *Supervisor*, Taunton; J. B. Gerould, North Attleboro'; J. B. Murphy, M. Perry, Taunton.

BRISTOL SOUTH.—Drs. S. W. Bowen, J. W. Coughlin, J. Gilbert, Fall River; A. M. Pierce, C. D. Prescott, *Supervisor*, New Bedford.

ESSEX NORTH.—Drs. C. E. Durant, *Supervisor*, Haverhill; H. G. Leslie, Amesbury; J. Pierce, Methuen; N. W. Pillsbury, Newburyport; L. J. Young, Haverhill.

ESSEX SOUTH.—Drs. F. F. Brigham, Lynn; J. H. Knowles, Gloucester; S. F. Quimby, *Supervisor*, Salem; G. A. Stickney, Beverly; F. E. Stone, Lynn.

FRANKLIN.—Drs. E. G. Best, Turner's Falls; F. J. Canedy, Shelburne Falls; C. G. Trow, Sunderland; A. C. Walker, *Supervisor*, Greenfield; W. M. Wright, Orange.

HAMPDEN.—Drs. E. B. Adams, *Supervisor*, Springfield; E. M. Davis, Holyoke; J. W. Hastings, Feeding Hills; E. H. Guild, G. L. Woods, Springfield.

HAMPSHIRE.—Drs. A. G. Blodgett, Ware; J. M. Fay, C. Seymour, *Supervisor*, G. D. Thayer, W. M. Trow, Northampton.

MIDDLESEX EAST.—Drs. H. G. Blake, J. H. Conway, Woburn; C. W. Harlow, Melrose; G. W. Nickerson, *Supervisor*, Stoneham; S. H. Parks, Reading.

MIDDLESEX NORTH.—Drs. W. Bass, T. F. Harrington, W. P. Lawler, T. G. McGannon, C. E. Simpson, *Supervisor*, Lowell.

MIDDLESEX SOUTH.—Drs. E. H. Bigelow, *Supervisor*, Framingham; F. G. Curtis, West Newton; H. B. McIntire, Cambridge; C. D. Sawin, Charlestown; H. A. Wood, Waltham.

NORFOLK.—Drs. C. A. Cheever, *Supervisor*, Dorchester; H. L. Chase, H. M. Cutts, Brookline; A. H. Hodgdon, Dedham; G. B. Stevens, Roxbury.

NORFOLK SOUTH.—Drs. W. A. Drake, North Weymouth; J. C. Fraser, *Supervisor*, East Weymouth; F. C. Granger, Randolph; N. S. Hunting, Quincy; J. W. Spooner, Hingham.

PLYMOUTH.—Drs. J. H. Averill, Campello; J. E. Bacon, H. F. Borden, W. P. Chisholm, Brockton; H. W. Dudley, *Supervisor*, Abington.

SUFFOLK.—Drs. J. B. Ayer, *Supervisor*, A. Coolidge, Jr., H. Jackson, J. C. Munro, H. Williams, Boston.

WORCESTER.—Drs. W. R. Gilman, E. V. Scribner, *Supervisor*, Worcester; C. G. Stearns, Leicester; G. L. Tobey, Clinton; L. F. Woodward, Worcester.

WORCESTER NORTH.—Drs. E. G. Cutter, *Supervisor*, Leominster; J. G. Henry, Winchendon; H. H. Lyons, A. P. Mason, Fitchburg; G. B. Underwood, West Gardner.

## Commissioners of Trials.

| | | |
|---|---|---|
| Barnstable | E. M. Parker | South Yarmouth. |
| Berkshire | A. T. Wakefield | Sheffield. |
| Bristol North | C. S. Holden | Attleboro'. |
| Bristol South | H. S. Swan | Middleboro'. |
| Essex North | F. A. Howe | Newburyport. |
| Essex South | J. Allen | Topsfield. |
| Franklin | C. Bowker | Bernardston. |
| Hampden | W. H. Bliss | Three Rivers. |
| Hampshire | F. Bonney | Hadley. |
| Middlesex East | J. S. Clark | Melrose. |
| Middlesex North | B. R. Benner | Lowell. |
| Middlesex South | A. Worcester | Waltham. |
| Norfolk | M. V. Pierce | Milton. |
| Norfolk South | N. S. Hunting | Quincy. |
| Plymouth | G. Osgood | Rockland. |
| Suffolk | F. W. Draper | Boston. |
| Worcester | C. H. Perry | Worcester. |
| Worcester North | F. W. Russell | Winchendon. |

## Officers of the District Medical Societies.

Barnstable.—Dr. W. S. Birge, Provincetown, *President*; Dr. Louis Edmonds, Harwich, *Vice-President;* Dr. J. H. Higgins, Marston's Mills, *Secretary;* Dr. G. N. Munsell, Harwich, *Treasurer.*

Berkshire.—Dr. W. W. Scofield, Dalton, *President;* Dr. M. L. Woodruff, Pittsfield, *Vice-President;* Dr. L. C. Swift, Pittsfield, *Secretary;* Dr. W. L. Paddock, Pittsfield, *Treasurer;* Dr. W. W. Leavitt, Pittsfield, *Librarian.*

Bristol North.—Dr. C. A. Atwood, Taunton, *President;* Dr. C. S. Holden, Attleboro', *Vice-President;* Dr. R. D. Dean, Taunton, *Secretary;* Dr. W. Y. Fox, Taunton, *Treasurer;* Dr. H. B. Baker, Dighton, *Librarian.*

Bristol South.—Dr. A. P. Webber, New Bedford, *President;* Dr. W. A. Dolan, Fall River, *Vice-President;* Dr. A. J. Abbe, Fall River, *Secretary and Treasurer.*

ESSEX NORTH.—Dr. J. J. Healy, Newburyport, *President;* Dr. F. B. Flanders, Lawrence, *Vice-President;* Dr. M. D. Clarke, Haverhill, *Secretary and Treasurer.*

ESSEX SOUTH.—Dr. J. E. Garland, Gloucester, *President;* Dr. H. W. Newhall, Lynn, *Vice-President;* Dr. A. N. Sargent, Salem, *Secretary;* Dr. G. Z. Goodell, Salem, *Treasurer;* Dr. J. E. Simpson, Salem, *Librarian.*

FRANKLIN.—Dr. M. P. Dole, Greenfield, *President;* Dr. F. H. Zabriskie, Greenfield, *Vice-President;* Dr. H. G. Stetson, Greenfield, *Secretary and Treasurer.*

HAMPDEN.—Dr. W. Holbrook, Palmer, *President;* Dr. L. J. Gibbs, Chicopee Falls, *Vice-President;* Dr. F. B. Sweet, Springfield, *Secretary and Treasurer.*

HAMPSHIRE.—Dr. H. H. Seelye, Amherst, *President;* Dr. J. A. Houston, Northampton, *Vice-President;* Dr. J. C. Fahey, Northampton, *Secretary;* Dr. A. H. Hoadley, Northampton, *Treasurer;* Dr. S. A. Clark, Northampton, *Librarian.*

MIDDLESEX EAST.—Dr. J. P. Bixby, North Woburn, *President;* Dr. C. E. Chase, Woburn, *Vice-President;* Dr. E. S. Jack, Melrose, *Secretary;* Dr. C. Dutton, Wakefield, *Treasurer.*

MIDDLESEX NORTH.—Dr. M. G. Parker, Lowell, *President;* Dr. F. W. Chadbourne, Lowell, *Vice-President;* Dr. W. G. Eaton, Lowell, *Secretary;* Dr. J. V. Meigs, Lowell, *Treasurer;* Dr. D. N. Patterson, Lowell, *Librarian.*

MIDDLESEX SOUTH.—Dr. E. H. Stevens, Cambridge, *President;* Dr. C. H. Cook, Natick, *Vice-President;* Dr. A. August, Cambridge, *Secretary;* Dr. W. Ela, Cambridge, *Treasurer;* Dr. F. H. Cleaves, Concord, *Librarian.*

NORFOLK.—Dr. E. P. Gerry, Jamaica Plain, *President;* Dr. F. W. Goss, Roxbury, *Vice-President;* Dr. J. C. D. Pigeon, Roxbury, *Secretary and Librarian;* Dr. E. G. Morse, Roxbury, *Treasurer.*

NORFOLK SOUTH.—Dr. G. W. Tinkham, Weymouth, *President;* Dr. J. C. Fraser, East Weymouth, *Vice-President;* Dr. J. F. Welch, Quincy, *Secretary, Treasurer and Librarian.*

PLYMOUTH.—Dr. E. D. Hill, Plymouth, *President;* Dr. A. A. MacKeen, Whitman, *Vice-President;* Dr. F. J. Ripley, Brock-

ton, *Secretary and Treasurer;* Dr. R. Hammond, Campello, *Librarian.*

SUFFOLK.—Dr. H. L. Burrell, Boston, *President;* Dr. T. M. Rotch, Boston, *Vice-President;* Dr. H. A. Lothrop, Boston, *Secretary;* Dr. A. S. Knight, Boston, *Treasurer;* Dr. B. J. Jeffries, Boston, *Librarian.*

WORCESTER.—Dr. W. Davis, Worcester, *President;* Dr. E. R. Wheeler, Spencer, *Vice-President;* Dr. L. C. Miller, Worcester, *Secretary;* Dr. G. O. Ward, Worcester, *Treasurer;* Dr. A. C. Getchell, Worcester, *Librarian.*

WORCESTER NORTH.—Dr. S. E. Greenwood, Templeton, *President;* Dr. C. H. Bailey, South Gardner, *Vice-President;* Dr. W. F. Sawyer, Fitchburg, *Secretary;* Dr. E. L. Fiske, Fitchburg, *Treasurer;* Dr. A. P. Mason, Fitchburg, *Librarian.*

# The Massachusetts Medical Society.

## PROCEEDINGS OF THE COUNCILLORS.

OCTOBER 4, 1899.

A STATED MEETING of the Councillors was held in the hall of the Medical Library Association, No. 19 Boylston Place, Boston, on Wednesday, October 4, 1899, at 11 o'clock, A.M.

The President, Dr. EDWIN B. HARVEY, in the chair.

The following Councillors were present:

*Barnstable.*
R. H. Faunce.

*Berkshire.*
J. F. A. Adams,
F. K. Paddock.

*Bristol North.*
F. A. Hubbard.

*Bristol South.*
G. de N. Hough.

*Essex North.*
I. J. Clarke,
J. A. Douglass,
J. F. Young.

*Essex South.*
H. K. Foster.

*Middlesex North.*
W. G. Eaton,
J. B. Field,
C. B. Sanders,
C. E. Simpson,
C. A. Viles.

*Middlesex South.*
Z. B. Adams,
E. H. Bigelow,
R. E. Brown,
J. E. Cleaves,
C. H. Cook,
J. G. Dearborn,
A. G. Griffin,
E. G. Hoitt,
E. D. Hooker,
R. P. Loring,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
C. E. Prior,
E. H. Stevens,
L. R. Stone,
H. P. Walcott,
A. C. Webber,
M. Wyman.

*Norfolk.*
H. D. Arnold,
J. S. Brownrigg,
J. E. Butler,
C. A. Cheever,
S. Crowell,
F. P. Emerson,
I. S. Fogg,
D. H. Fuller,
G. W. Kaan,
E. G. Morse,
L. F. Vickery.

*Plymouth.*
B. F. Hastings,
A. E. Paine,
F. G. Wheatley.

*Suffolk.*
J. B. Ayer,

E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
B. F. Campbell,
D. W. Cheever,
E. G. Cutler,
T. W. Fisher,
R. H. Fitz,
G. W. Gay,
C. M. Green,
J. Homans,
F. I. Knight,
A. B. Morong,
C. B. Porter,
J. M. Putnam,
M. H. Richardson,
W. L. Richardson,
T. M. Rotch,
G. H. M. Rowe,
F. C. Shattuck,
G. B. Shattuck,
A. M. Sumner,
G. G. Tarbell,
H. F. Vickery,
J. C. White,
E. N. Whittier.

*Worcester.*
W. P. Bowers,
W. Davis,
G. E. Francis,
E. B. Harvey,
S. P. Holbrook,
J. O. Marble,
E. W. Norwood,
H. M. Quinby,
E. V. Scribner.

*Worcester North.*
L. G. Chandler,
B. H. Hartwell.

Total, 88.

The record of the previous meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

*Vermont.*—Drs. L. G. Chandler, of Townsend; R. W. Greene, of Worcester.

*New York.*—Drs. H. Colt, of Pittsfield; J. C. Hubbard, of Holyoke.

*New York State Medical Association.*—Drs. R. T. Edes, of Jamaica Plain; E. G. Cutler, of Boston.

The Committee on Membership and Finance reported through Dr. Stone. In accordance with their recommendation the following were allowed to resign:

Chauncy Adams, of Concord, N. H.
George W. Cutler, of Waltham.
George W. Holden, of Denver, Col.
James A. Keown, of Lynn.
Isaac Louis, of Boston.
Henry P. Lovewell, of Providence, R. I.

Also the following was allowed to retire:

J. Langdon Sullivan, of Malden.

Also the following, having failed to pay their assessment dues, were deprived of the privileges of membership under By-Law VIII.:

Charles E. Adams, of Santa Clara, Cal.
Henry W. Brown, of Binghamton, N. Y.

Lewis N. Bump, of Somerville.
Edwin H. Codding.
William L. Faxon, of Middleborough.
George W. Gates, of San Francisco, Cal.
Charles A. Gould, of Newton Highlands.
Samuel K. Harriman, of Providence, R. I.
Patrick H. Keefe, of Providence, R. I.
Horace M. Locke, of Sturbridge.
Albert N. Mehrenlender, of New York, N. Y.
James W. Moore, of New York, N. Y.
Laurentius M. Nason, of Worcester.
Julius Selva, of Coainto, Nicaragua.
Harry J. Thompson, of W. Philadelphia, Pa.
James N. Tobin, of Pittsfield.
Melville E. Webb, of Boston.

*Voted*, That the vote passed at the Councillors' meeting, May 29, 1866, as printed among the Standing Votes and Resolves, "That the fee for the engraved diploma of Fellowship be fixed at five dollars," be rescinded, and that the issuing of the diploma cease from this date.

Adjourned at 11.25 A.M.

FRANCIS W. GOSS,
*Recording Secretary.*

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FEBRUARY 7, 1900.

A STATED MEETING of the Councillors was held in the hall of the Medical Library Association, No. 19 Boylston Place, Boston, on Wednesday, February 7, 1900, at 12 o'clock, noon.

The President, Dr. EDWIN B. HARVEY, in the chair.

The following Councillors were present:

| *Berkshire.* | *Essex North.* | *Essex South.* |
|---|---|---|
| F. K. Paddock. | C. G. Carleton, | H. K. Foster, |
| | I. J. Clarke, | A. F. Hunt. |
| *Bristol South.* | J. A. Douglass, | |
| J. H. Gifford, | J. F. Richards, | *Hampden.* |
| G. de N. Hough, | O. Warren, | F. Holyoke, |
| J. H. Jackson. | J. F. Young. | C. W. Jackson. |

*Middlesex East.*
F. W. Graves,
J. M. Harlow.

*Middlesex North.*
W. G. Eaton,
J. B. Field,
C. B. Sanders.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
R. A. Blood,
J. E. Cleaves,
C. H. Cook,
E. Cowles,
J. G. Dearborn,
E. D. Hooker,
R. P. Loring,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
G. A. Oviatt,
G. C. Pierce,
E. H. Stevens,
L. R. Stone,
H. P. Walcott,
A. C. Webber,
J. W. Willis.

*Norfolk.*
H. D. Arnold,
J. S. Brownrigg,
J. E. Butler,
C. A. Cheever,
H. M. Cutts,
D. G. Eldridge,
I. S. Fogg,
I. H. Hazelton,
G. H. Ingalls,
G. W. Kaan,
E. B. Lane,
E. G. Morse,
J. J. Sewall,
L. F. Vickery.

*Norfolk South.*
J. F. Welch.

*Plymouth.*
H. W. Dudley,
B. F. Hastings,
N. K. Noyes,
A. E. Paine,
F. G. Wheatley.

*Suffolk.*
J. B. Ayer,
E. M. Buckingham,
J. F. Bush,
B. F. Campbell,
D. W. Cheever,
A. Coolidge, Jr.,
E. G. Cutler,
F. W. Draper,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
W. H. H. Hastings,
B. J. Jeffries,
A. H. Nichols,
C. B. Porter,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
F. C. Shattuck,
G. B. Shattuck,
A. M. Sumner,
G. G. Tarbell,
H. F. Vickery.

*Worcester.*
W. P. Bowers,
G. E. Francis,
E. B. Harvey,
S. P. Holbrook,
J. O. Marble,
E. W. Norwood,
H. M. Quinby,
E. V. Scribner,
E. R. Wheeler,
L. Wheeler.

*Worcester North.*
L. G. Chandler,
B. H. Hartwell,
F. H. Thompson.

Total, 94.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

*Maine.*—Drs. E. N. Whittier, of Boston; J. J. Healy, of Newburyport.

*New Hampshire.*—Drs. C. C. Odlin, of Melrose; B. H. Hartwell, of Ayer.

*Rhode Island.*—Drs. W. Davis, of Worcester; J. M. French, of Milford.

*Connecticut.*—Drs. J. M. Fay, of Northampton; W. H. Pomeroy, of Springfield.

*New Jersey.*—Drs. D. W. Cheever, of Boston; J. L. Hildreth, of Cambridge.

*Convention for Revision of U. S. Pharmacopœia.*—Drs. R. T. Edes, B. F. Davenport, F. G. Wheatley.

The following Committee was appointed:

*To Audit the Treasurer's Accounts.*—Drs. H. M. Cutts, H. F. Vickery.

The Committee on Membership and Finance reported through Dr. Draper. In accordance with their recommendation it was voted that the following be allowed to resign:

Edward J. Cutter, of Leominster.
Henry A. Deane, of East Windsor, Conn.
Paul Plummer, of Collinsville, Conn.
William H. Russell, of Ipswich.
George P. Sprague, of Lexington, Ky.

Also, that the following be placed on the retired list;

Rollin H. Phelps, of Littleton.

Also, that the following be deprived of the privileges of membership in accordance with By-Law VIII. for non-payment of assessment dues:

Abraham I. Dinenson, of Boston.
Charles Mackin, of Milford.

The following amendments to the By-Laws, edition of 1897, were offered by the Secretary:

By-Law I., on page 9, shall be amended by striking out the words "possesses a diploma, or its equivalent," and substituting the words, "has received a diploma"; so that the clause shall read: "has received a diploma from such school."

By-Law II., on page 9, shall be amended by striking out its entire text and inserting in the place thereof the following:

II. A candidate, found to possess the qualifications prescribed, shall become a Fellow of the Society on paying the assessment for the current year and subscribing to the By-Laws within the three months next following such finding, and not otherwise.

By-Law XII., on page 17, shall be amended by striking out lines 12 to 18 inclusive, and inserting in the place thereof the following:

The Secretary of the District Society shall see that all the requirements of the By-Laws are complied with by each successful candidate for Fellowship, and shall thereupon promptly report his name and address to the Recording Secretary of The Massachusetts Medical Society.

After remarks in support thereof by Dr. Draper, it was

*Voted*, That the amendments be reported to the Society with the recommendation that they be adopted.

Dr. Folsom presented a communication favoring the recommendation of the State Board of Insanity for the passage of an Act for the State Care of the Insane. He moved, and it was voted, that a Committee of three be appointed by the Chair to appear before the Legislature and advocate such an Act.

The following were appointed to constitute the Committee:

Drs. C. F. Folsom, J. C. White, D. W. Cheever.

Adjourned at 12.50 P.M.

FRANCIS W. GOSS,
*Recording Secretary.*

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JUNE 12, 1900.

THE ANNUAL MEETING of the Councillors was held in Paul Revere Hall, Mechanic Building, Boston, on Tuesday, June 12, 1900, at 5 o'clock, P.M.

The President, Dr. EDWIN B. HARVEY, in the chair.

The following Councillors were present:

*Barnstable.*
H. S. Kelley, Jr.,
F. A. Rogers.

*Berkshire.*
J. F. A. Adams,
O. J. Brown,
H. Bushnell,
F. K. Paddock.

*Bristol North.*
A. M. Round.

*Bristol South.*
G. de N. Hough,
J. H. Jackson.

*Essex North.*
C. G. Carleton,
J. J. Clarke,
J. G. McAllister,
J. F. Young.

*Essex South.*
W. W. Eaton,
C. W. Haddock,
A. F. Hunt,
P. Mooney.

*Franklin.*
F. J. Canedy.

*Hampden.*
D. E. Keefe,
S. A. Mahoney,
G. L. Woods.

*Middlesex East.*
H. G. Blake,
C. Dutton,
J. W. Heath.

*Middlesex North.*
T. G. McGannon,
C. E. Simpson.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
R. E. Brown,
J. E. Cleaves,
C. H. Cook,
E. Cowles,
J. G. Dearborn,
E. G. Hoitt,
E. D. Hooker,
R. P. Loring,
H. P. Makechnie,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
G. A. Oviatt,
C. E. Prior,
E. H. Stevens,
L. R. Stone,
H. P. Walcott,
A. C. Webber,
J. W. Willis.

*Norfolk.*
H. D. Arnold,
F. A. Bragg,
J. S. Brownrigg,
J. E. Butler,
C. A. Cheever,
F. C. Dunbar,
D. G. Eldridge,
F. P. Emerson,
D. H. Fuller,
I. H. Hazelton,
H. R. Hitchcock,
G. H. Ingalls,
G. W. Kaan,
E. B. Lane,
E. G. Morse,
H. V. Reynolds,
L. F. Vickery.

*Norfolk South.*
J. F. Welch.

*Plymouth.*
H. W. Dudley,
B. F. Hastings,
A. E. Paine,
F. G. Wheatley.

*Suffolk.*
E. H. Bradford,
E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
B. F. Campbell,
D. W. Cheever,
A. Coolidge, Jr.,
H. Derby,
F. W. Draper,
T. W. Fisher,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
J. Homans,
F. I. Knight,
A. L. Mason,
A. B. Morong,
A. H. Nichols,
J. J. Putnam,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
F. C. Shattuck,
G. B. Shattuck,
J. B. Swift,
G. G. Tarbell,
H. F. Vickery,
J. C. Warren,
J. C. White,
E. N. Whittier.

*Worcester.*
P. P. Comey,
G. E. Francis,
E. B. Harvey,
S. P. Holbrook,
E. W. Norwood,
E. V. Scribner,
E. R. Wheeler,
L. Wheeler,
A. Wood.

*Worcester North.*
L. G. Chandler,
B. H. Hartwell,
F. H. Thompson.

Total, 112.

The record of the last meeting was read and accepted.

The Secretary read the names of new and of deceased Fellows.

The Treasurer, Dr. Buckingham, read his annual report.

The Auditing Committee reported that they found the

accounts correctly cast, and that the Treasurer has in his possession the securities called for.

*Voted*, To accept the Treasurer's report.

The Committee on Membership and Finance reported through Dr. Draper and recommended that $3,500.00 of the surplus in the treasury be distributed among the District Societies.

*Voted*, That the recommendation be adopted.

In accordance with the recommendation of the same Committee it was voted, that the following be allowed to resign:

Hairabed S Jelalian, of Boston.
Mary Sanderson, of Worcester.
Eugene P. Stone, of United States Navy.
Elsie B. Taylor, of Boston.
Herbert Warren, of Worcester.

Also, that the following be placed on the retired list:

Daniel A. Cleaveland, of Hyannis.
James R. Deane, of Newton Highlands.
Samuel L. Dutton, of Newton Centre.
Thomas H. Gage, of Worcester.
William Ingalls, of Roxbury.
Gustav Liebmann, of Boston.
Albert B. Robinson, of Roxbury.
Alonzo L. Stickney, of Ashburnham.
Oscar F. Swasey, of Beverly.
Peleg Wadsworth, of Malden.
John W. Willis, of Waltham.

The Committee on Publications presented their report.

The Librarian, Dr. Brigham, presented his report.

The Committee on Nominations reported a list of candidates for the offices of the Society for the ensuing year, and the same were elected by ballot:

| | |
|---|---|
| *President* . . | Dr. FRANK W. DRAPER, of Boston. |
| *Vice-President* . | Dr. WILLIAM W. EATON, of Danvers. |
| *Treasurer* . . | Dr. EDWARD M. BUCKINGHAM, of Boston. |
| *Corresponding Sec'y* | Dr. CHARLES W. SWAN, of Brookline. |
| *Recording Secretary* | Dr. FRANCIS W. GOSS, of Roxbury. |
| *Librarian* . . | Dr. EDWIN H. BRIGHAM, of Brookline. |

Dr. CHARLES F. FOLSOM, of Boston, was chosen Orator for the annual meeting of the Society in 1901.

[Dr. Folsom having declined, the President with the Committee of Arrangements subsequently appointed Dr. GEORGE E. FRANCIS, of Worcester, Orator for 1901.]

*Voted*, That the next annual meeting of the Society be held in Boston on the second Wednesday in June, 1901.

The following Standing Committees were appointed:

*Of Arrangements.*

M. Storer, W. H. Prescott, J. C. Hubbard,
S. Crowell, F. G. Balch, Farrar Cobb.

*On Publications.*

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

*On Membership and Finance.*

E. G. Cutler, L. R. Stone, F. W. Goss,
W. Ela, C. M. Green.

*To Procure Scientific Papers.*

H. L. Burrell, A. K. Stone, F. H. Thompson,
G. de N. Hough, H. W. Van Allen.

*On Ethics and Discipline.*

G. E. Francis, F. C. Shattuck, C. G. Carleton,
E. Cowles, J. F. A. Adams.

*On Medical Diplomas.*

H. E. Marion, E. N. Whittier, O. F. Rogers.

*On State and National Legislation.*

F. W. Draper, H. P. Bowditch, S. D. Presbrey,
S. W. Abbott, B. H. Hartwell.

Adjourned at 6.15 P.M.

FRANCIS W. GOSS,
*Recording Secretary.*

# The Massachusetts Medical Society.

## PROCEEDINGS OF THE SOCIETY.

### ANNUAL MEETING.

FIRST DAY.

JUNE 12, 1900.

SECTIONAL meetings were held in Mechanic Building, Boston, on Tuesday, June 12, 1900, at 2 o'clock, P.M.

The sections were organized and papers were presented as follows:

SECTION IN MEDICINE.

Dr. FREDERICK C. SHATTUCK, of Boston . Chairman.
Dr. JAMES J. MINOT, of Boston . . . . Secretary.

VARIATIONS IN THE CLINICAL COURSE OF PNEUMONIA, ACCORDING TO THE BACTERIAL ETIOLOGY.—By Dr. Frederick H. Baker, of Worcester.

LOCAL AND GENERAL TREATMENT IN UNCOMPLICATED SEVERE CASES OF PNEUMONIA. SPECIAL CONSIDERATION OF THE USE OF ALCOHOL AND OTHER STIMULANTS IN PNEUMONIA. DELIRIUM AND DELIRIUM TREMENS AS COMPLICATIONS OF PNEUMONIA.—By Dr. Norman F. Chandler, of Medford.

PECULIARITIES IN PNEUMONIA OF CHILDREN AND ELDERLY PEOPLE.—By Dr. Alfreda B. Withington, of Pittsfield.

PROGNOSIS OF PNEUMONIA, AND TREATMENT OF THE FAMILY.—By Dr. James W. Hannum, of Ludlow.

CONTAGIOUSNESS OF PNEUMONIA.—By Dr. John N. Cooledge, of Boston.

DELAYED AND PROLONGED RESOLUTION IN PNEUMONIA. SPECIAL TREATMENT. PROGNOSIS.—By Dr. Stephen A. Mahoney, of Holyoke.

SERUMTHERAPY IN PNEUMONIA.—By Dr. William H. Smith, of Boston.

HYDROTHERAPY IN PNEUMONIA.—By Dr. Simon Baruch, of New York City.

SECTION IN SURGERY.

Dr. WALTER P. BOWERS, of Clinton . . . Chairman.
Dr. FRANKLIN G. BALCH, of Boston . . . Secretary.

CARBOLIC GANGRENE.—By Dr. Francis B. Harrington, of Boston.

SUPPURATIVE PERICARDITIS.—By Dr. Charles B. Porter, of Boston.

THE TREATMENT OF RETRO-DISPLACEMENTS OF THE UTERUS.—By Dr. Charles H. Hare, of Boston.

THE TREATMENT OF PROLAPSE OF THE UTERUS.—By Dr. Walter L. Burrage, of Boston.

THE OPERATIVE TREATMENT OF UTERINE DISPLACEMENTS BY VAGINAL FIXATION.—By Dr. William N. Swift, of New Bedford.

At 8 o'clock, P.M., the Shattuck Lecture was delivered by Dr. WILLIAM H. WELCH, of Baltimore, Md.

Following the lecture a reception was given by the Society to its officers.

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## SECOND DAY.

### JUNE 13, 1900.

THE Society met in Mechanic Building, Boston, on Wednesday, June 13, 1900, at 9 o'clock, A.M., for the exercises of the one hundreth and nineteenth Anniversary.

The President, Dr. EDWIN B. HARVEY, in the chair.

The record of the last annual meeting was read and accepted.

The Secretary read the names of Fellows reported admitted since the last annual meeting, and of those whose deaths had been recorded.

*Admissions reported since June 13, 1899.*

| | | |
|---|---|---|
| 1900 | Abbott, Edson Moses | Woburn. |
| 1900 | Alfred, James | Brockton. |
| 1899 | Andrews, Harold Virgil | Boston. |
| 1899 | Atkinson, Roger Trowbridge | Boston. |
| 1899 | Bailey, William Thomas | Boston. |
| 1899 | Baker, Lewis Forrester | Fitchburg. |
| 1900 | Baketel, Harrie Sheridan | Melrose Highlands. |
| 1899 | Barnes, Harry Aldrich | Boston. |
| 1900 | Barrell, Charles Lowell | Boston. |
| 1900 | Barton, John Alfred | Fitchburg. |
| 1899 | Beckley, Chester Charles | Lancaster. |
| 1899 | Birge, Russell Hall | Cleveland, Ohio. |
| 1900 | Black, George Luke | Lowell. |
| 1899 | Bridgman, Burt Nicholas | Jamaica Plain. |
| 1899 | Briggs, Lloyd Vernon | Boston. |
| 1899 | Brown, Alphonso Bickford | Newburyport. |
| 1899 | Brown, Anthony Leopold | Springfield. |
| 1899 | Buck, Maurice Allen | Billerica. |
| 1899 | Burke, William Henry | Cambridge. |
| 1899 | Burleigh, Robert Fletcher | South Braintree. |
| 1899 | Burns, Frederick Stanford | Boston. |
| 1899 | Buttler, Charles Voorhees | Worcester. |
| 1900 | Cahill, John Thomas | Lawrence. |
| 1900 | Callahan, Joseph Thomas | Woburn. |
| 1900 | Carothers, William Harsha | Melrose. |
| 1899 | Carroll, John Philip | Woburn. |
| 1900 | Cate, Frederick Symon | Melrose. |
| 1899 | Chapman, Charles Ratchford | Springfield. |
| 1900 | Charteris, Mary Alena | Worcester. |
| 1900 | Chase, Edwin Llewellyn | Shrewsbury. |
| 1900 | Choate, Horace Henry | Salem. |
| 1900 | Clap, Edmund Wright | Boston. |
| 1900 | Cone, Dwight Eleazer | Fall River. |
| 1900 | Crane, Carl Custer | Melrose. |
| 1899 | Crawford, Francis Xavier | South Boston. |
| 1900 | Crittenden, Samuel Wright | Mattapan. |
| 1900 | Cronin, Jeremiah Augustine | South Boston. |
| 1899 | Crowley, Ambrose | Cambridge. |
| 1900 | Culbert, Robert Ronayne | Roxbury. |
| 1900 | Cummings, Frederic Russell | Boston. |
| 1900 | Currier, William Eugene | Boston. |
| 1899 | Dalton, Martin James | Melrose. |
| 1900 | Daudelin, Simeon Alphonse | Worcester. |
| 1899 | Davis, Charles Henry | Beverly. |
| 1899 | Davis, Lincoln | Boston. |
| 1900 | Dennett, Alonzo Gustin | Lowell. |

| | | |
|---|---|---|
| 1900 | Dodge, Arthur Malcolm | Boston. |
| 1899 | Dole, Charles Frederick | South Boston. |
| 1900 | Dorgan, Joseph Aloysius | Lawrence. |
| 1900 | Dorman, Albert Barnes | Winthrop. |
| 1900 | Duckering, William West | Dorchester. |
| 1900 | Easton, Elwood Tracy | Boston. |
| 1900 | Eaton, Lilley | Winchester. |
| 1900 | Elliot, Henry Libbey | Salem. |
| 1900 | Elliott, Richard Andrew | Avon. |
| 1899 | Ely, Richard Skinner | West Townsend. |
| 1900 | Emery, George Edwin | Worcester. |
| 1899 | Estabrook, Charles Taylor | Worcester. |
| 1900 | Finch, George Hardy | Springfield. |
| 1899 | Flynn, John Leo | Cambridge. |
| 1900 | French, Charles Ephraim | Brookline. |
| 1900 | Golden, Lazarus | Boston. |
| 1900 | Goodwin, Harold Carl | Tewksbury. |
| 1899 | Greenough, Clara Mary | Deerfield. |
| 1900 | Haines, Ignatius | Cambridge. |
| 1899 | Hammond, William John | Dorchester. |
| 1899 | Handy, Harry Tucker | Jamaica Plain. |
| 1900 | Hart, Henry Brown | East Dennis. |
| 1899 | Haskins, Frank Henry | Springfield. |
| 1899 | Hatch, Edward Sparhawk | Brighton. |
| 1899 | Hathaway, John Gael | New Bedford. |
| 1900 | Hazlett, Olive Binkley | South Framingham. |
| 1900 | Heald, Charles Gerry | Pepperell. |
| 1900 | Heaton, Thomas Henry | Cambridge. |
| 1900 | Hixon, Edwin Colfax | Cambridge. |
| 1900 | Holmes, Harry Bigelow | Adams. |
| 1900 | Hopkins, Bertrand Hiram | Ayer. |
| 1899 | Howard, Joseph Francis | Lawrence. |
| 1900 | Howell, Thomas | Worcester. |
| 1900 | Hurd, Albert Gordon | Millbury. |
| 1899 | Hurd, Randolph Campbell | Newburyport. |
| 1899 | Jackson, William Andrew, Jr. | Haydenville. |
| 1899 | Jaques, Louis Désiré Onésiphoré | Worcester. |
| 1900 | Kinney, William D'Arcy | Osterville. |
| 1899 | Knight, Charles Eugene | Rockland. |
| 1899 | Knowlton, John Greenleaf Whittier | Boston. |
| 1900 | Ladd, Maynard | Boston. |
| 1899 | Laighton, Florence Marion | New York, N. Y. |
| 1899 | LaMotte, Henry | Northampton. |
| 1899 | Latham, Benoni Mowry | Mansfield. |
| 1899 | Lawrence, Nellie Louise | Roxbury. |
| 1900 | Leslie, Herbert Granville | Amesbury. |
| 1900 | Livingston, Ernest George | Lowell. |

| | | |
|---|---|---|
| 1899 | Lougee, William Wheeler | Malden. |
| 1900 | Lowell, Martha Eleanor | Roxbury. |
| 1899 | Lynch, Charles Francis | Springfield. |
| 1900 | McDermott, Joseph Edward | Boston. |
| 1899 | MacDonald, William Joseph | Charlestown. |
| 1900 | McEvoy, George Albert | Roxbury. |
| 1899 | McIntosh, John David | Rockland. |
| 1900 | McMurray, Francis Michael | Fitchburg. |
| 1899 | Mains, Charles Frederick | Dorchester. |
| 1900 | Masten, Charles Howard | Worcester. |
| 1899 | Merriam, Franklin Henry | Sterling. |
| 1900 | Milot, Wilfrid François | Taunton. |
| 1900 | Morin, Jacob Mauritz | Brockton. |
| 1900 | Murphy, Edward Vincent | Fall River. |
| 1899 | Nason, Arthur Clark | Newburyport. |
| 1900 | Nelson, John William | Waltham. |
| 1899 | Newton, Aaron Lewis | Northfield. |
| 1900 | Nicola, Charles Chesterfield | South Lancaster. |
| 1899 | Nielsen, Edwin Björne | West Newton. |
| 1899 | Northrop, Clarence Clark | Boston. |
| 1900 | Norton, George Paul | Fitchburg. |
| 1899 | Noyes, Henry Allen | Pittsfield. |
| 1900 | O'Brien, Thomas James | South Boston. |
| 1899 | O'Connor, John Edward | Fall River. |
| 1900 | O'Donnell, James Coughlin | Lowell. |
| 1899 | Ohnesorg, Karl | U. S. Navy. |
| 1900 | Patterson, William Francis | Charlestown. |
| 1899 | Paul, Luther Gordon | Boston. |
| 1900 | Peck, Luke Baker | Brookline. |
| 1899 | Perry, Edgar | Boston. |
| 1900 | Pillsbury, Boyden Harlin | Lowell. |
| 1899 | Place, Ralph Waldo | Boston. |
| 1900 | Prevaux, John Jacob | Haverhill. |
| 1900 | Purvis, Alice Hatheway | Roxbury. |
| 1900 | Randall, George Merrill | Lowell. |
| 1900 | Riley, Elizabeth Angela | Roxbury. |
| 1899 | Russell, Simon James | Springfield. |
| 1899 | Sayward, William Henry, Jr. | Dorchester. |
| 1900 | Schmidt, Frederick Sextus | Jamaica Plain. |
| 1900 | Schorer, Cornelia Bernhardine Johanna | Boston. |
| 1900 | Sears, Stephen Hull | Yarmouthport. |
| 1899 | Sharp, Walter Nevin | Saxonville. |
| 1900 | Smith, Frank Herbert | Hadley. |
| 1899 | Smith, Harry Atherton | West Roxbury. |
| 1899 | Snow, Frederick Stedman | Roxbury. |
| 1900 | Soutter, Robert | Boston. |
| 1899 | Spooner, Henry Garrettson | Boston. |

| | | |
|---|---|---|
| 1899 | Stevens, Ralph Emerson | Marlborough. |
| 1899 | Stockwell, Edgar Washburn | New Marlborough. |
| 1899 | Stockwell, Herbert Emmons | Stockbridge. |
| 1899 | Stoddard, Mortimer James | Springfield. |
| 1900 | Stone, Warren Buxton | New Bedford. |
| 1900 | Street, Lionel Alexander Burnet | Brookline. |
| 1900 | Swain, Howard Townsend | Boston. |
| 1900 | Symonds, Alice Gertrude | Haverhill. |
| 1899 | Thorning, William Burton | Winchendon. |
| 1899 | Tilden, Irving Niles | New Bedford. |
| 1900 | Upham, Emily Clark | Boston. |
| 1899 | von Groll, Maximilian Charles | Roxbury. |
| 1899 | Washburn, Frank Hall | Lynn. |
| 1900 | Wells, Abner Toothaker | Jamaica Plain. |
| 1900 | Wheeler, Lucia Anna | Worcester. |
| 1900 | Wilkins, George Clarence | South Boston. |
| 1899 | Williams, Hugh | Boston. |
| 1899 | Woodbury, Herbert Elwell | Natick. |
| 1899 | Woods, Charles Edwin | Lunenburg. |

Total, 159.

## *List of Deceased Fellows.*

| Admitted. | Name. | Residence. | Date of Death. | Age. |
|---|---|---|---|---|
| 1878 | Bartlett, George Pinkham | Woburn | March 27, 1900 | 49 |
| 1873 | Brechin, William Pitt | Boston | Dec. 10, 1899 | 48 |
| 1852 | Bronson, John Richardson | Attleborough | May 9, 1900 | 70 |
| 1852 | Chamberlain, Cyrus Nathaniel | Andover | July 18, 1889 | 70 |
| 1852 | Deane, Adam Calhoun | Greenfield | Nov. 7, 1899 | 76 |
| 1885 | French, George Morrill | Malden | Feb. 23, 1900 | 45 |
| 1854 | Gilbert, John Henry | Quincy | Aug. 3, 1899 | 72 |
| 1878 | Graves, Frank Walker | Woburn | March 13, 1900 | 57 |
| 1854 | Greene, Francis Coles | Easthampton | Aug. 2, 1899 | 72 |
| 1895 | Hall, Milton Wilder | Roxbury. | Jan. 9, 1900 | 51 |
| 1870 | Hastings, William Henry Howe | Boston | Feb. 16, 1900 | 60 |
| 1864 | Hutchins, Isaiah | West Acton | Aug. 7, 1899 | 69 |
| 1854 | Jenks, Thomas Leighton | Boston | Oct. 31, 1899 | 69 |
| 1883 | Kemble, Lawrence Grafton | Salem | Aug. 8, 1899 | 37 |
| 1897 | Kingsley, Charles | Marlborough | Dec. 29, 1899 | 30 |
| 1864 | McLaughlin, James Augustin | Boston | Sept. 8, 1899 | 85 |
| 1839 | Marsh, Austin | Carlisle | Feb. 2, 1900 | 88 |
| 1846 | Morse, Luther Blodgett | Watertown | May 26, 1900 | 79 |
| 1891 | O'Hearn, William Henry | Lawrence | June 4, 1900 | 30 |
| 1882 | *Paget, Sir James | London, Eng | Dec. 30, 1899 | 85 |
| 1854 | Perkins, Edward Augustus | Boston | Feb. 20, 1900 | 72 |
| 1874 | Pierce, Gardner Carpenter | Ashland | May 18, 1900 | 62 |
| 1865 | Plunkett, Francis Charles | Lowell | Nov. 29, 1899 | 57 |
| 1869 | Stackpole, Paul Augustine | Dover, N. H. | Nov. 28, 1900 | 86 |
| 1862 | Street, Charles Carroll | Boston | May 7, 1900 | 64 |
| 1881 | Sturgis, Russell, Jr. | Boston | July 17, 1899 | 42 |
| 1851 | Warren, George Washington | West Boylston | April 22, 1900 | 81 |
| 1888 | Woodbridge, Luther Dana | Williamstown | Nov. 3, 1899 | 48 |
| 1880 | Wright, Walter Melvin | Orange | June 24, 1899 | 53 |

* Honorary.

Total, 29.

The Treasurer's report was read.

Amendments to the By-Laws (see Proceedings, pages 45 and 46), were presented from the Councillors with their recommendation that they be adopted.

*Voted*, That these amendments be adopted, and that the By-Laws be changed as indicated.

Papers were read as follows:

*Diseases of Joints often Diagnosed as Rheumatic, or Complicated with Rheumatism.*

TRAUMATIC JOINTS. (SPRAINS AND BRUISES).—By Dr. Homer Gage, of Worcester.

SEPTIC AND GONORRHŒA JOINTS.—By Dr. Charles A. Porter, of Boston.

RHEUMATISM. (ACUTE ARTICULAR).—By Dr. Ernest S. Jack, of Melrose.

NEUROPHATIC JOINTS.—By Dr. Sidney A. Lord of Boston.

ARTHRITIS DEFORMANS. (OSTEOID, RHEUMATOID).—By Dr. Joel E. Goldthwait, of Boston.

TO WHAT EXTENT DOES RHEUMATIC AND GOUTY DIATHESIS ENTER INTO THE FOREGOING CONDITIONS AS AN ETIOLOGICAL FACTOR? WHAT IS THE SCIENTIFIC BASIS FOR SUCH A TERM? —By Dr. William H. Porter, of New York City.

The following delegates from other State Medical Societies were present:

*New Hampshire.*—Dr. R. F. Oliver.
*Rhode Island.*—Dr. H. G. Palmer.
*Connecticut.*—Dr. H. G. Howe.

Drs. Palmer and Howe presented the greetings of their respective Societies.

At 12 o'clock the Annual Discourse was delivered by Dr. ARTHUR T. CABOT, of Boston.

*Voted*, That the thanks of the Society be presented to the Orator for his earnest, thoughtful and instructive address.

At 1 P.M. the Annual Dinner was served to more than one thousand Fellows.

FRANCIS W. GOSS,
*Recording Secretary.*

# TREASURER'S REPORT.

APRIL 15, 1900.

There has been no change in the invested funds. They are as follows:

| | | |
|---|---|---|
| COTTING FUND— | | |
| Deposit Roxbury Institution for Savings | $1000 00 | |
| " Provident " " " | 1000 00 | |
| " Suffolk Savings Bank . . | 1000 00 | |
| | $3,000 00 | $3,000 00 |
| PHILLIPS FUND— | | |
| United States 4 per cent. bond . | | 10,000 00 |
| SHATTUCK FUND— | | |
| Annuity Policy Mass. Hosp. Life Ins. Co. | | 9,166 87 |
| PERMANENT FUND— | | |
| Annuity Policy Mass. Hosp. Life Ins. Co. | $7,542 81 | |
| " " " " " " " | 2,079 87 | |
| " " " " " " " | 1,630 62 | |
| Massachusetts 3 1-2 per cent. bonds . | 2,000 00 | |
| Deposit Franklin Savings Bank . . | 1,074 48 | |
| | $14,327 78 | $14,327 78 |
| | | $36,494 65 |

The report of the General Fund is as follows:—

*Receipts.*

| | | | |
|---|---|---|---|
| Balance, April 15, 1899 . . . . . | | | $8,901 27 |
| Assessments— | | | |
| Paid to Treasurer at Annual Meeting . | | $765 | |
| Paid to Treasurer at other times . . | | 370 | |
| Paid to District Treasurers:— | | | |
| Barnstable . . . | $100 | | |
| Berkshire . . . . | 245 | | |
| Bristol North . . | 170 | | |
| Bristol South . . . | 315 | | |
| Essex North . . . | 380 | | |
| Essex South . . . | 525 | | |
| Franklin . . . | 115 | | |
| Hampden . . . . | 565 | | |
| Hampshire . . . | 235 | | |
| Middlesex East . . . | 235 | | |
| Middlesex North . . | 445 | | |
| Middlesex South . . | 1,240 | | |
| Norfolk . . . . | 1,395 | | |
| Norfolk South . . . | 180 | | |
| *Amounts carried forward,* | $6,145 | $1,135 | $8,901 27 |

| | | | |
|---|---|---|---|
| *Amounts brought forward,* | $6,145 | $1,135 00 | $8,901 27 |
| Plymouth | 200 | | |
| Suffolk | 2,295 | | |
| Worcester | 810 | | |
| Worcester North | 190 | | |
| | $9,640 | | |
| Total collected in Districts | | $9,640 00 | |
| Total assessments | | $10,775 00 | $10,775 00 |
| Premium on foreign check | | | .10 |
| Over-payment by check | | | .05 |
| Income— | | | |
| From annuities Mass. Hosp. Life Ins. Co. | | 816 79 | |
| Interest U. S. bond | | 400 00 | |
| " Mass. bonds | | 70 00 | |
| " Savings Bank deposits | | 143 82 | |
| " Deposit Mt. Vernon Nat. Bank | | 125 38 | |
| " " Bay State Trust Co. | | 62 74 | |
| | | $1,618 73 | |
| | | | $1,618 73 |
| Sale of extra dinner tickets | | 8 00 | |
| Fee for engraved diploma | | 5 00 | |
| Total | | $21,308 15 | $21,308 15 |

*Expenses.*

| | | | |
|---|---|---|---|
| Treasurer :— | | | |
| Internal revenue stamps | 1 72 | | |
| Stationery, stamped envelopes, printing | 52 14 | | |
| Card catalogue case and cards | 15 00 | | |
| Box at Bay State Trust Co. | 10 00 | | |
| Clerical work at annual meeting | 30 00 | | |
| | $108 86 | 108 86 | |
| Secretary :— | | | |
| Stationery, printing, engrossing and clerical work, postage | | 283 84 | |
| Librarian :— | | | |
| Stationery, delivering Communications and postage | | 222 77 | |
| Custodian :— | | | |
| Making record boxes | | 21 50 | |
| Censors | | 571 15 | |
| Supervisors' mileage | | 7 10 | |
| District Treasurers:— | | | |
| Commissions and expenses | | 544 40 | |
| Committee On Publications :— | | | |
| Printing | | 733 13 | |
| *Amount carried forward,* | | $2,492 75 | |

| | | |
|---|---|---|
| *Amount brought forward,* | | $2,492 75 |
| Committee on Instruction in Physiology and Hygiene :— | | |
| Printing and clerical work | | 59 74 |
| Committee on Papers :— | | |
| Printing and clerical work | | 33 69 |
| Committee on Legislation :— | | |
| Printing | | 3 50 |
| Legal expenses | | 315 00 |
| Catalogue:— | | |
| Printing, binding, clerical work, delivery | | 727 52 |
| Shattuck Lecture | | 201 25 |
| Cotting Lunch | | 141 90 |
| Annual Meeting :— | | |
| Carriages | 7 00 | |
| Stationery | 52 15 | |
| Stereopticon work | 22 50 | |
| Music and flowers | 90 00 | |
| Towel Supply Co. | 9 25 | |
| Mechanics Hall | 603 85 | |
| Platforms | 81 00 | |
| Card signs | 8 20 | |
| Stenographer | 10 00 | |
| Police | 8 29 | |
| Caterer | 1,786 81 | |
| Cigars | 96 00 | |
| Clerical work | 16 50 | |
| Elevated Railway excursion | 15 65 | |
| Badges | 3 63 | |
| Incidentals | 16 10 | |
| | $2,826 93 | 2,826 93 |
| Rent | | 150 00 |
| Salaries | | |
| Librarian | 400 00 | |
| Secretary | 500 00 | |
| Treasurer | 500 00 | |
| | $1,400 00 | $1,400 00 |
| Annual Dividend :— | | |
| Barnstable | 39 40 | |
| Berkshire | 97 34 | |
| Bristol North | 64 90 | |
| Bristol South | 111 24 | |
| Essex North | 157 59 | |
| Essex South | 201 62 | |
| Franklin | 41 72 | |
| Hampden | 201 62 | |
| Hampshire | 92 70 | |
| Middlesex East | 97 34 | |
| Middlesex North | 173 81 | |
| *Amounts carried forward,* | $1,279 28 | $8,352 28 |

| | | | |
|---|---|---|---|
| *Amounts brought forward,* | $1,279 28 | $8,352 28 | |
| Middlesex South | 551 55 | | |
| Norfolk | 560 82 | | |
| Norfolk South | 83 47 | | |
| Plymouth | 99 66 | | |
| Suffolk | 1,038 20 | | |
| Worcester | 319 81 | | |
| Worcester North | 67 21 | | |
| | $4,000 00 | 4,000 00 | |
| Total | | $12,352 28 | 12,352 98 |
| Balance, April 15, 1900 | | | $8,955 27 |

EDWARD M. BUCKINGHAM,
*Treasurer.*

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The undersigned, a duly appointed Committee, having examined the books of the Treasurer, find the accounts properly cast and that he has in his possession the securities called for.

HARRY M. CUTTS.
HERMAN F. VICKERY.

MAY 26, 1900.

# Officers of The Massachusetts Medical Society.

1900—1901.

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CHOSEN JUNE 12, 1900.

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| | | |
|---|---|---|
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| EDWARD M. BUCKINGHAM, | Boston, . | TREASURER. |
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| FRANCIS W. GOSS, . . . . | Roxbury, | REC. SECRETARY. |
| EDWIN H. BRIGHAM, . . . | Brookline, | LIBRARIAN. |

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*On Membership and Finance.*

E. G. CUTLER, L. R. STONE, F. W. GOSS,
W. ELA, C. M. GREEN.

*To Procure Scientific Papers.*

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G. DE N. HOUGH, H. W. VAN ALLEN.

*On Ethics and Discipline.*

G. E. FRANCIS, F. C. SHATTUCK, C. G. CARLETON,
E. COWLES, J. F. A. ADAMS.

*On Medical Diplomas.*

H. E. MARION, E. N. WHITTIER, O. F. ROGERS.

*On State and National Legislation.*

F. W. DRAPER, H. P. BOWDITCH, S. D. PRESBREY,
S. W. ABBOTT, B. H. HARTWELL.

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[Arranged according to seniority.]

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G. W. Tinkham,
F. W. Chadbourne,
J. P. Bixby,
H. L. Burrell,
C. H. Cook,
F. B. Flanders,
A. A. MacKeen,
C. H. Bailey,
J. A. Houston,
C. A. Atwood,
L. J. Gibbs,
G. A. Stickney,
W. A. Dolan,
W. W. Scofield,
M. P. Dole,
L. Edmonds.

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Bristol South.—Drs. J. T. Bullard, New Bedford; G. L. Ellis, Middleboro'; J. H. Gifford, J. H. Jackson, Fall River; G. de N. Hough, C. D. Prescott, New Bedford.

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PLYMOUTH.—Drs. H. W. Dudley, Abington; B. F. Hastings, Whitman; N. K. Noyes, Duxbury; A. E. Paine, Brockton, F. G. Wheatley, North Adams.

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WORCESTER NORTH.—Drs. L. G. Chandler, Townsend; B. H. Hartwell, Ayer; C. H. Rice, F. H. Thompson, Fitchburg.

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BRISTOL SOUTH.—Drs. S. W. Bowen, J. W. Coughlin, W. T. Learned, Fall River; A. M. Pierce, C. D. Prescott, *Supervisor*, New Bedford.

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ESSEX SOUTH.—Drs. F. F. Brigham, Lynn; H. K. Foster, *Supervisor*, Peabody; J. H. Knowles, Gloucester; S. F. Quimby, Salem; F. E. Stone, Lynn.

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HAMPDEN.—Drs. E. M. Davis, F. Holyoke, Holyoke; J. W. Hastings, Feeding Hills; P. Kilroy, G. L. Woods, *Supervisor*, Springfield.

HAMPSHIRE.—Drs. A. G. Blodgett, Ware; J. G. Hayes, Williamsburg; J. M. Fay, *Supervisor*, G. D. Thayer, W. M. Trow, Northampton.

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MIDDLESEX SOUTH.—Drs. F. G. Curtis, West Newton; H. B. McIntire, Cambridge; C. D. Sawin, Charlestown; J. Tolman, Arlington; H. A. Wood, *Supervisor*, Waltham.

NORFOLK.—Drs. H. M. Cutts, *Supervisor*, Brookline; C. G. Dewey, Dorchester; G. H. Francis, Brookline; H. B. Hitchcock, Hyde Park; G. B. Stevens, Roxbury.

NORFOLK SOUTH.—Drs. W. A. Drake, North Weymouth; J. C. Fraser, *Supervisor*, East Weymouth; F. C. Granger, Randolph; N. S. Hunting, Quincy; J. W. Spooner, Hingham.

PLYMOUTH.—Drs. J. H. Averill, Campello; J. E. Bacon, H. F. Borden, W. P. Chisholm, Brockton; H. W. Dudley, *Supervisor*, Abington.

SUFFOLK.—Drs. J. B. Ayer, *Supervisor*, A. Coolidge, Jr., H. Jackson, J. C. Munro, G. H. Washburn, Boston.

WORCESTER.—Drs. O. H. Everett, H. Gage, E. V. Scribner, *Supervisor*, Worcester; C. L. French, Clinton; C. G. Stearns, Leicester.

Worcester North.—Drs. J. G. Henry, Winchendon; H. H. Lyons, A. P. Mason, F. H. Thompson, *Supervisor*, Fitchburg; G. B. Underwood, West Gardner.

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| | | |
|---|---|---|
| Barnstable | A. T. Walker | Falmouth. |
| Berkshire | A. T. Wakefield | Sheffield. |
| Bristol North | F. A. Hubbard | Taunton. |
| Bristol South | H. S. Swan | Middleboro'. |
| Essex North | F. A. Howe | Newburyport. |
| Essex South | J. Allen | Topsfield. |
| Franklyn | C. Bowker | Bernardston. |
| Hampden | A. O. Squire | Springfield. |
| Hampshire | F. Bonney | Hadley. |
| Middlesex East | J. S. Clark | Melrose. |
| Middlesex North | B. R. Benner | Lowell. |
| Middlesex South | A. Worcester | Waltham. |
| Norfolk | M. V. Pierce | Milton. |
| Norfolk South | N. S. Hunting | Quincy. |
| Plymouth | G. Osgood | Rockland. |
| Suffolk | F. W. Draper | Boston. |
| Worcester | C. H. Perry | Worcester. |
| Worcester North | A. W. Sidney | Fitchburg. |

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BRISTOL SOUTH.—Dr. W. A. Dolan, Fall River, *President;* Dr. M. H. Leonard, New Bedford, *Vice-President;* Dr. A. J. Abbe, Fall River, *Secretary and Treasurer.*

ESSEX NORTH.—Dr. F. B. Flanders, Lawrence, *President;* Dr. J. F. Croston, Haverhill, *Vice-President;* Dr. M. D. Clarke, Haverhill, *Secretary and Treasurer.*

ESSEX SOUTH.—Dr. G. A. Stickney, Beverly, *President;* Dr. C. E. Meader, Lynn, *Vice-President;* Dr. B. F. Sturgis, Jr., Salem, *Secretary;* Dr. G. Z. Goodell, Salem, *Treasurer;* Dr. G. C. Littlefield, Salem, *Librarian.*

FRANKLIN.—Dr. M. P. Dole, Greenfield, *President;* Dr. F. H. Zabriskie, Greenfield, *Vice-President;* Dr. H. G. Stetson, Greenfield, *Secretary and Treasurer.*

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HAMPSHIRE.—Dr. J. A. Houston, Northampton, *President;* Dr. C. R. Gardner, Northampton, *Vice-President;* Dr. J. C. Fahey, Northampton, *Secretary;* Dr. A. H. Hoadley, Northampton, *Treasurer;* Dr. S. A. Clark, Northampton, *Librarian.*

MIDDLESEX EAST.—Dr. J. P. Bixby, North Woburn, *President;* Dr. C. E. Chase, Woburn, *Vice-President;* Dr. E. S. Jack, Melrose, *Secretary;* Dr. C. Dutton, Wakefield, *Treasurer.*

MIDDLESEX NORTH.—Dr. F. W. Chadbourne, Lowell, *President;* Dr. W. H. Lathrop, Lowell, *Vice-President;* Dr. W. G. Eaton, Lowell, *Secretary;* Dr. J. V. Meigs, Lowell, *Treasurer;* Dr. D. N. Patterson, Lowell, *Librarian.*

MIDDLESEX SOUTH.—Dr. C. H. Cook, Natick, *President;* Dr. E. G. Hoitt, Marlboro', *Vice-President;* Dr. A. August, Cambridge, *Secretary;* Dr. W. Ela, Cambridge, *Treasurer;* Dr. E. S. Dow, Allston, *Librarian.*

NORFOLK.—Dr. F. W. Goss, Roxbury, *President;* Dr. C. F. Withington, Roxbury, *Vice-President;* Dr. J. C. D. Pigeon, Roxbury, *Secretary and Librarian;* Dr. E. G. Morse, Roxbury, *Treasurer.*

NORFOLK SOUTH.—Dr. G. W. Tinkham, Weymouth, *President;* Dr. J. C. Fraser, East Weymouth, *Vice-President;* Dr. S. W. Ellsworth, Quincy, *Secretary, Treasurer and Librarian.*

PLYMOUTH.—Dr. A. A. MacKeen, Whitman, *President;* Dr. R. Hammond, Campello, *Vice-President;* Dr. F. H. Burnett, Brockton, *Secretary and Treasurer;* Dr. W. H. Adams, East Bridgewater, *Librarian.*

SUFFOLK.—Dr. H. L. Burrell, Boston, *President;* Dr. T. M. Rotch, Boston, *Vice-President;* Dr. H. A. Lothrop, Boston, *Secretary;* Dr. W. H. Prescott, Boston, *Treasurer;* Dr. B. J. Jeffries, Boston, *Librarian.*

WORCESTER.—Dr. W. Davis, Worcester, *President;* Dr. E. R. Wheeler, Spencer, *Vice-President;* Dr. L. C. Miller, Worcester, *Secretary;* Dr. G. O. Ward, Worcester, *Treasurer;* Dr. A. C. Getchell, Worcester, *Librarian.*

WORCESTER NORTH.—Dr. C. H. Bailey, South Gardner, *President;* Dr. H. W. Lyons, Fitchburg, *Vice-President;* Dr. W. F. Sawyer, Fitchburg, *Secretary;* Dr. E. L. Fiske, Fitchburg, *Treasurer;* Dr. A. P. Mason, Fitchburg, *Librarian.*

# The Massachusetts Medical Society.

## PROCEEDINGS OF THE COUNCILLORS.

OCTOBER 3, 1900.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, October 3, 1900, at 12 o'clock, noon.

The President, Dr. FRANK W. DRAPER, in the chair.

The following Councillors were present:

*Berkshire.*
O. J. Brown,
H. Bushnell.

*Bristol North.*
S. D. Presbrey.

*Bristol South.*
J. H. Gifford,
G. deN. Hough.

*Essex North.*
I. J. Clarke,
J. G. McAllister.

*Essex South.*
A. F. Hunt,
C. E. Meader.

*Hampden.*
G. L. Woods.

*Middlesex East.*
C. Dutton,
J. W. Heath.

*Middlesex North.*
W. G. Eaton,
J. B. Field,
C. A. Viles.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
J. E. Cleaves,
O. W. Collins,
C. H. Cook,
J. G. Dearborn,
E. D. Hooker,
H. P. Makechnie,
J. T. G. Nichols,
A. L. Norris,
C. E. Prior,
F. M. Sherman,
L. R. Stone,
H. P. Walcott,
A. C. Webber,
J. W. Willis,
H. A. Wood.

*Norfolk.*
H. D. Arnold,
F. A. Bragg,
J. S. Brownrigg,
J. E. Butler,
F. P. Emerson,
D. H. Fuller,
I. H. Hazelton,
G. W. Kaan,
E. G. Morse.

*Plymouth.*
H. W. Dudley,
B. F. Hastings,
N. K. Noyes,
A. E. Paine.

*Suffolk.*
J. B. Ayer,
H. L. Burrell,
J. F. Bush,
B. F. Campbell,
D. W. Cheever,
A. Coolidge, Jr.,
E. G. Cutler,

F. W. Draper,
R. H. Fitz,
G. W. Gay,
C. M. Green,
B. J. Jeffries,
F. I. Knight,
J. H. McCollom,
A. H. Nichols,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
G. H. M. Rowe,
F. C. Shattuck,
G. B. Shattuck,
J. B. Swift,
J. C. White,
E. N. Whittier,
H. Williams.

*Worcester.*
G. E. Francis,
E. B. Harvey,
E. V. Scribner,
L. Wheeler.

*Worcester North.*
B. H. Hartwell,
C. H. Rice,
F. H. Thompson.

Total, 77.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

*Vermont.*—Drs. E. P. Miller, of Fitchburg; G. C. McClean, of Springfield.

*New York.*—Drs. L. Wheeler, of Worcester; E. O. Otis, of Boston.

*New York State Medical Association.*—Drs. J. L. Hildreth, of Cambridge; L. C. Swift, of Pittsfield.

*Voted*, That Edmund Randolph Peaslee Fourtin, of Waltham, be restored to the privileges of membership.

Adjourned at 12.35 P.M.

FRANCIS W. GOSS,
*Recording Secretary.*

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FEBRUARY 6, 1901.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, February 6, 1901, at 12 o'clock, noon.

The President, Dr. FRANK W. DRAPER, in the chair.

The following Councillors were present:

*Bristol South.*
G. deN. Hough.

*Essex North.*
I. J. Clarke,
F. A. Howe,
J. G. McAllister,
J. F. Young.

*Essex South.*
H. K. Foster,
P. Mooney.

*Hampden.*
G. L. Woods.

*Middlesex North.*
W. G. Eaton,
M. G. Parker.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
R. E. Brown,
J. E. Cleaves,
O. W. Collins,
C. H. Cook,
E. Cowles,
A. G. Griffin,
E. D. Hooker,
H. P. Makechnie,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
G. A. Oviatt,
C. E. Prior,
L. R. Stone,
A. C. Webber,
J. W. Willis,
M. Wyman.

*Norfolk.*
H. D. Arnold,
E. E. Bancroft,
J. S. Brownrigg,
J. E. Butler,
S. Crowell,
H. M. Cutts,
F. P. Denny,
D. G. Eldridge,
F. P. Emerson,
D. H. Fuller,
I. H. Hazelton,
G. W. Kaan,
E. N. Libby,
E. G. Morse.

*Plymouth.*
H. W. Dudley,
B. F. Hastings,
A. E. Paine,
F. G. Wheatley.

*Suffolk.*
J. B. Ayer,
E. M. Buckingham,
J. F. Bush,
D. W. Cheever,
A. Coolidge, Jr.,
E. G. Cutler,
H. Derby,
F. W. Draper,
R. H. Fitz,
C. F. Folsom,
M. F. Gavin,
C. M. Green,
J. Homans,
F. I. Knight,
A. L. Mason,
J. H. McCollom,
A. B. Morong,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
G. H. M. Rowe,
J. B. Swift,
H. F. Vickery,
J. C. Warren,
J. C. White,
E. N. Whittier,
H. Williams.

*Worcester.*
E. B. Harvey,
S. P. Holbrook,
J. O. Marble,
E. W. Norwood,
E. V. Scribner,
L. Wheeler.

*Worcester North.*
B. H. Hartwell,
F. H. Thompson.

Total, 82.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

*Maine.* — Drs. L. Huntress, of Lowell; L. J. Gibbs, of Chicopee Falls.

*New Hampshire.* — Drs. S. W. Abbott, of Newton; F. B. Stephenson, of Portsmouth, N. H.

*Rhode Island.*—Drs. J. W. Spooner, of Hingham; G. deN. Hough, of New Bedford.

*Connecticut.*—Drs. J. O. Marble, of Worcester; D. E. Keefe, of Springfield.

*New Jersey.*—Drs. M. H. Richardson, of Boston; R. H. Faunce, of Sandwich.

The following Committee was appointed:

*To Audit the Treasurer's Accounts.*—Drs. H. F. Vickery, H. W. Broughton.

The Committee on Membership and Finance reported through Dr. Cutler. In accordance with their recommendation it was voted that the following be allowed to resign:

Lyman Allen, of Burlington, Vt.
John E. Bradbury, of Denver, Col.
Wencelas J. B. Chagnon, of St. Hyacinthe, P. Q.
Luther H. Gulick, of Brooklyn, N. Y.
Augustus S. Knight, of New York, N. Y.
John E. McKenty, of New York, N. Y.
Frederick C. Page, of Woodstock, Ill.
John E. Pratt, of Dumont, N. J.
Will H. Swan, of Colorado Springs, Col.

Also, that the following be placed on the retired list:

Charles E. Vaughan, of Santa Barbara, Cal.

Also, that the following be deprived of the privileges of membership in accordance with By-Law VIII. for non-payment of assessment dues:

Herman J. Achard, of Roselle, Ill.
James Burton, of Gilbertville.
Charles A. Davenport, of Newton.
Frank H. Drew.
Joseph E. Duxbury, of Boston.
George W. Greenleaf.
Perry E. Joslin, of Hopkinton.
Edward D. Laforce, of Central Falls, R. I.
Albert C. Leach, of Orange.
John C. McCarthy, of Malden.
Robert J. Mansfield of Springfield.
Jonathan H. Meigs, of Beachmont.
Frank O. Mosher.
Charles H. Quirk, of Buenos Ayres, S. A.
Gilman P. Robinson, of Atlanta, Ga.
Alexander Rovinski, of Boston.
Albert A. Sargent, The Klondike.
Whitmell P. Small, of Great Barrington.
James S. Sullivan, of Randolph.

Voted, That the following be restored to the privileges of membership in the Society:

Christopher Philip Lyons, of Boston.
George Wheelock Grover, of Sheffield.

Adjourned at 12.50 P.M.

FRANCIS W. GOSS,
*Recording Secretary.*

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JUNE 11, 1901.

THE ANNUAL MEETING of the Councillors was held in the Medical Library, Boston, on Tuesday, June 11, 1901, at 5 o'clock, P.M.

The President, Dr. FRANK W. DRAPER, in the chair.

The following Councillors were present:

*Barnstable.*
C. E. Harris.

*Berkshire.*
J. F. A. Adams,
O. J. Brown,
H. Bushnell,
F. K. Paddock.

*Bristol North.*
C. A. Atwood,
S. D. Presbrey,
T. J. Robinson.

*Bristol South.*
W. A. Dolan,
J. H. Gifford,
G. deN. Hough,
J. H. Jackson.

*Essex North.*
J. G. McAllister,
J. F. Young.

*Essex South.*
W. W. Eaton,
A. F. Hunt,
C. E. Meader.

*Franklin.*
G. P. Twitchell.

*Hampden.*
J. E. Marsh.

*Hampshire.*
C. F. Branch,
C. A. Byrne,
G. D. Thayer.

*Middlesex East.*
J. P. Bixby.

*Middlesex North.*
G. A. Harlow,
J. E. Lamoureux.

*Middlesex South.*
Z. B. Adams,
D. E. Baker,
R. E. Brown,
J. E. Cleaves,
C. H. Cook,
E. Cowles,
A. G. Griffin,
E. A. Hobbs,
E. G. Hoitt,
E. D. Hooker,
R. P. Loring,
H. B. McIntire,
H. P. Makechnie,
H. E. Marion,
J. T. G. Nichols,
A. L. Norris,
G. A. Oviatt,
F. M. Sherman,
E. H. Stevens,
L. R. Stone,
H. P. Walcott,

A. C. Webber,
J. W. Willis.

*Norfolk.*

F. R. Abbe,
F. A. Bragg,
J. E. Butler,
S. Crowell,
H. M. Cutts,
F. P. Denny,
F. C. Dunbar,
F. P. Emerson,
I. S. Fogg,
G. H. French,
I. H. Hazelton,
H. R. Hitchcock,
A. D. Hodges,
G. H. Ingalls,
G. W. Kaan,
R. M. Merrick,
E. G. Morse,
L. F. Vickery,
A. G. Williams.

*Norfolk South.*

C. S. Adams,
N. S. Hunting,
E. N. Mayberry.

*Plymouth.*

H. W. Dudley,
A. E. Paine,
F. J. Ripley,
F. G. Wheatley.

*Suffolk.*

J. B. Ayer,
E. M. Buckingham,
J. F. Bush,
B. F. Campbell,
D. W. Cheever,
A. Coolidge, Jr.,
E. G. Cutler,
H. Derby,
F. W. Draper,
T. W. Fisher,
R. H. Fitz,
C. F. Folsom,
W. W. Gannett,
C. M. Green,
J. Homans,
F. I. Knight,
J. H. McCollom,
A. H. Nichols,
C. B. Porter,
A. Post,
J. J. Putnam,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
F. C. Shattuck,
G. B. Shattuck,
H. F. Vickery,
J. C. White,
E. N. Whittier,
G. Wolcott.

*Worcester.*

W. Davis,
O. H. Everett,
G. E. Francis,
E. B. Harvey,
S. P. Holbrook,
J. O. Marble,
E. W. Norwood,
H. M. Quinby,
W. G. Reed,
E. R. Wheeler,
L. Wheeler,
A. Wood.

*Worcester North.*

B. H. Hartwell,
C. H. Rice,
F. H. Thompson.

Total, 119.

The record of the last meeting was read and accepted.

The Secretary read the names of new and of deceased Fellows.

The Treasurer, Dr. Buckingham, read his annual report.

The Auditing Committee reported that they found the accounts correctly cast, and that the Treasurer has in his possession the securities called for.

*Voted*, To accept the Treasurer's report.

The Committee on Membership and Finance reported through Dr. Cutler, and recommended that $4,000.00 of the surplus in the treasury be distributed among the District Societies.

*Voted,* That the recommendation be adopted.

In accordance with the recommendation of the same Committee it was voted, that the following be allowed to resign:

George W. Chamberlain, of Springfield.
Edward M. Child, of Brooklyn, N. Y.
Alice L. Cobb, of Florence, Italy.
Frank A. Dunbar, of Bonita, Cal.
John D. McIntosh, of Rockland.
Richard M. Pearce, of Philadelphia, Pa.
Katherine F. Peckham, of Putnam Heights, Conn.
George H. Thomas, of Kansas City, Mo.
Gertrude W. VanPelt, of Point Loma, Cal.

Also, that the following be placed on the retired list:

Robert F. Andrews, of Gardner.
John J. Colton, of Lowell.
Susan E. Crocker of Boston.
Thomas H. Dearing, of Braintree.
Samuel A. Green, of Boston.
George N. Munsell, of Harwich.
Augustus C. Walker, of Greenfield.

Also, that the following be deprived of the privileges of membership under By-Law VIII.:

Alphonso V. Bowker, of Athol.
Edward O. Robinson.

The Committee on Publications presented their report.

The Librarian, Dr. Brigham, presented his report.

The Committee on Nominations reported a list of candidates for the offices of the Society for the ensuing year, and the same were elected by ballot:

| | |
|---|---|
| *President* . . | Dr. FRANK W. DRAPER, of Boston. |
| *Vice-President* . | Dr. AUGUSTUS C. WALKER, of Greenfield. |
| *Treasurer* . . | Dr. EDWARD M. BUCKINGHAM, of Boston. |
| *Corresponding Sec'y* | Dr. CHARLES W. SWAN, of Brookline. |
| *Recording Secretary* | Dr. FRANCIS W. GOSS, of Roxbury. |
| *Librarian* . . | Dr. EDWIN H. BRIGHAM, of Brookline. |

Dr. GEORGE S. EDDY, of Fall River, was chosen Orator for the annual meeting of the Society in 1902.

*Voted*, That the next annual meeting of the Society be held in Boston on the second Wednesday in June, 1902.

The following Standing Committees were appointed:

*Of Arrangements.*

S. Crowell, F. G. Balch, Farrar Cobb,
W. H. Prescott, J. C. Hubbard, A. P. Perry.

*On Publications.*

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

*On Membership and Finance.*

E. G. Cutler, L. R. Stone, F. W. Goss,
W. Ela, C. M. Green.

*To Procure Scientific Papers.*

A. K. Stone, C. F. Withington, W. H. Pomeroy,
J. B. Blake, G. L. Richards.

*On Ethics and Discipline.*

G. E. Francis, F. C. Shattuck, C. G. Carleton,
E. Cowles, J. F. A. Adams.

*On Medical Diplomas.*

H. E. Marion, E. N. Whittier, O. F. Rogers.

*On State and National Legislation.*

F. W. Draper, H. P. Bowditch, S. D. Presbrey,
S. W. Abbott, B. H. Hartwell.

The following were elected to fill vacancies:

Censors for Hampden District, F. Holyoke, W. M. E. Mellen.
Censor for Middlesex East District, M. A. Cummings.
Councillor for Suffolk District, O. F. Wadsworth.

Adjourned at 6.20 P.M.

FRANCIS W. GOSS,
*Recording Secretary.*

# The Massachusetts Medical Society.

## PROCEEDINGS OF THE SOCIETY.

### ANNUAL MEETING.

FIRST DAY.

JUNE 11, 1901.

SECTIONAL meetings were held in the Medical Library, Boston, on Tuesday, June 11, 1901, at 2 o'clock, P.M.

The sections were organized and papers were presented as follows:

SECTION IN MEDICINE.

Dr. WILLIAM W. EATON, of Danvers . . Chairman.
Dr. MAYNARD LADD, of Boston . . . . Secretary.

*Subject.—The Diseases of the Nutrition of Infants.*

GENERAL CONSIDERATIONS.—By Dr. T. M. Rotch, of Boston.

INFANTILE ATROPHY.—By Dr. J. L. Morse, of Boston.

INFANTILE SCURVY.—By Dr. E. L. Peirson, of Salem.

RHACHITIS.—By Dr. A. R. Crandell, of Taunton.

ASSOCIATION OF ANÆMIA WITH CHRONIC ENLARGEMENT OF THE SPLEEN.—By Dr. A. H. Wentworth, of Boston.

CRETINISM.—By Dr. C. S. Millet, of Brockton.

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THE PUPIL AS AN INDEX OF DISEASE.—By Dr. E. E. Jack, of Boston.

SECTION IN SURGERY.

Dr. JOHN C. IRISH, of Lowell . . . . . Chairman.
Dr. JOHN T. BOTTOMLEY, of Boston . . . Secretary.

*Subject.—In what cases of Disease of the Alimentary Tract, not Emergencies, should Operations be advised either for Relief or Cure?*

STRICTURE OF THE ŒSOPHAGUS.—By Dr. Theodore Dunham, of New York City.

GASTRIC ULCER.—By Dr. A. T. Cabot, of Boston.

GASTRIC CANCER.—By Dr. W. M. Conant, of Boston.

GALL STONES.—By Dr. M. H. Richardson, of Boston.

INTESTINAL CANCER.—By Dr. F. B. Lund, of Boston.

OBSTRUCTIVE DISEASE OF THE LOWER BOWEL.—By Dr. H. O. Marcy, of Boston.

At 8 o'clock, P.M., the Shattuck Lecture was delivered by Dr. WILLIAM F. WHITNEY, of Boston.

Following the lecture a reception was given by the Society to its Officers.

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## SECOND DAY.

### JUNE 12, 1901.

THE Society met in Chickering Hall, Boston, on Wednesday, June 12, 1901, at 9.30 o'clock, A.M., for the exercises of the one hundreth and twentieth Anniversary.

The President, Dr. FRANK W. DRAPER, in the chair.

The record of the last annual meeting was read and accepted.

The Secretary read the names of Fellows reported admitted since the last annual meeting, and of those whose deaths had been recorded.

*Admissions reported since June 12, 1900.*

| | | |
|---|---|---|
| 1900 | Aldrich, Albert Clinton | Somerville. |
| 1901 | Allen, Freeman | Boston. |
| 1900 | Atwood, Charles Fenner | Hyde Park. |
| 1900 | Bacon, Newton Samuel | Cambridge. |
| 1901 | Balmer, William Edward | Boston. |
| 1900 | Barnes, James Arthur | Worcester. |
| 1900 | Bedell, Charles Ellsworth | Somerville. |
| 1901 | Beebe, Theodore Chapin, Jr. | Boston. |
| 1901 | Beede, M Josephine | Boston. |
| 1901 | Bertrand, Alexis Evariste | Lowell. |
| 1901 | Bicknell, Ralph Emerson | North Weymouth. |
| 1900 | Blair, George Kenniston | Salem. |
| 1901 | Blake, Le Grand | Milford. |
| 1900 | Bolster, Augustus Sargent | Worcester. |
| 1900 | Boutelle, Harry Clifton | Dorchester. |
| 1901 | Boyer, Joseph Napoleon | Springfield. |
| 1901 | Bryant, Fred | Worcester. |
| 1901 | Burke, Walter Thomas | Boston. |
| 1901 | Burnham, Melvin Page | Tewksbury. |
| 1901 | Clark, Edward James | Lowell. |
| 1901 | Clark, Ezra Warren | Brockton. |
| 1900 | Connolly, John Matthew | Dorchester. |
| 1901 | Coolidge, Sumner | Watertown. |
| 1900 | Cornwell, Herbert Cerdá de Vilarrestau | Cambridge. |
| 1900 | Creesy, Everett Larcom | Beverly. |
| 1901 | Cushing, Adelaide Olga | Roxbury. |
| 1899 | Davis, Percy Guy | Deerfield. |
| 1901 | DeGrandpré, Louis Philippe | Worcester. |
| 1901 | Denning, William Edward | Worcester. |
| 1901 | Donlan, Charles Edwin | Lowell. |
| 1901 | Doucett, Frederick Luke | East Weymouth. |
| 1901 | Dresser, Arthur James | Tewksbury. |
| 1901 | Drummond, Edward Augustine | Providence, R. I. |
| 1901 | Eastman, Alexander Crane | Boston. |
| 1900 | Elmere, John Alfred | Boston. |
| 1900 | Emerson, William Robie Patten | Boston. |
| 1900 | Ewing, George Winburn | Middleton. |
| 1900 | Ferguson, Edward Hugh | Dorchester. |
| 1901 | Ferguson, Robert Henry | Dorchester. |
| 1901 | Forster, Robert William | Lawrence. |
| 1901 | Fraser, Margaret Ethel Victoria | Boston. |
| 1901 | Friedman, Leo Victor | Boston. |
| 1901 | Galvin, William | Blackinton. |

| | | |
|---|---|---|
| 1900 | Gardner, Harrie Milton | Athol. |
| 1900 | Gay, William Frederick | Boston. |
| 1901 | Germain, Harry Hosmer | Boston. |
| 1901 | Goodrich, Harriet Rawson | Salem. |
| 1901 | Gray, Alice Maud | Dorchester. |
| 1901 | Greene, Daniel Crosby, Jr. | Boston. |
| 1901 | Hale, Willis Le Baron | Attleboro' Falls. |
| 1901 | Hall, James Frank | Lowell. |
| 1900 | Hall, Rufus Hacker | Everett. |
| 1900 | Harding, Walter Allen | Everett. |
| 1901 | Harriman, Charles Henry | Whitinsville. |
| 1900 | Harris, William DeBlois | Lynn. |
| 1900 | Hawes, Alfred Taylor | Lynn. |
| 1901 | Hazen, Robert | Boston. |
| 1901 | Healy, Daniel Laurence | South Framingham. |
| 1901 | Hill, George Sumner | Brookline. |
| 1900 | Hill, Hibbert Winslow | Jamaica Plain. |
| 1900 | Holt, Frank Hammett | Boston. |
| 1901 | Howard, Frederick Hollis | Williamstown. |
| 1900 | Howell, William Westcott | West Roxbury. |
| 1901 | Hubbard, George William | South Hadley. |
| 1901 | Jack, Lewis Harlow | Boston. |
| 1901 | Johnson, David Joseph | Roxbury. |
| 1901 | Johnson, Walter Sydney | Boston. |
| 1901 | Jouett, Fred Robert | Cambridge. |
| 1900 | Kepler, Charles Ober | Boston. |
| 1901 | King, James Aldrich | Lynn. |
| 1900 | Kingsbury, Charles Henry | Oxford. |
| 1901 | Lambert, Fred DeForest | Salem. |
| 1900 | Lane, Walter Appleton | Milton. |
| 1901 | Lawrence, Arthur Abbott | Milton. |
| 1900 | Legg, Arthur Thornton | Chelsea. |
| 1901 | Lewis, George Fred | New Bedford. |
| 1900 | McCormick, Alfred Hugh | Northampton. |
| 1900 | McGee, Fannie Maria | Waban. |
| 1900 | McGirr, Felix Francis | Cambridge. |
| 1901 | McLeod, John Scott | Roxbury. |
| 1900 | McNair, Robert Hamilton | Longmeadow. |
| 1900 | McNiff, William Joseph | East Pepperell. |
| 1901 | Mandell, Augustus Hamlin | New Bedford. |
| 1901 | Marcy, Henry Orlando, Jr. | Boston. |
| 1900 | Merritt, Victor Sulviro | Haydenville. |
| 1900 | Miller, Jared Homer | North Brookfield. |
| 1900 | Miller, Samuel Osgood | Three Rivers. |
| 1901 | Mitchell, William | Highlandville. |
| 1900 | Murphy, Charles Oscar | Roxbury. |

| | | |
|---|---|---|
| 1900 | Murray, Charles Albert | Cambridge. |
| 1901 | Newton, LeRoy Allan | Greenfield. |
| 1901 | Nicola, Mary Byington | South Lancaster. |
| 1900 | Nightingale, James | Worcester. |
| 1901 | O'Connell, Delia Maria | Roxbury. |
| 1901 | Odiorne, Walter Burlingame | Boston. |
| 1900 | Orr, Samuel Sanford | Weston. |
| 1901 | Osgood, Robert Bayley | Boston. |
| 1900 | Peggs, Harry MacDonald | Somerville. |
| 1901 | Peterson, John Adna | Hingham. |
| 1900 | Pettigrew, Richard Richardson | Roslindale. |
| 1901 | Pike, Winfred Carle | Dorchester. |
| 1901 | Pitta, João Carlos daSilva | New Bedford. |
| 1900 | Pomeroy, Harris Starr | Peabody. |
| 1901 | Potter, Alexander Carleton | Boston. |
| 1900 | Provandie, Paul Hector | Melrose Highlands |
| 1900 | Reilly, James Aloysius | South Boston. |
| 1900 | Rich, Edwin Willis | Cambridge. |
| 1901 | Rosa, Henry Antonio | Fall River. |
| 1900 | Rust, Frank Lee Drummond | Boston. |
| 1901 | Ryder, Charles Edward | South Boston. |
| 1901 | Ryder, George Hale | Boston. |
| 1901 | Saunders, Joseph Henry | Brookline. |
| 1901 | Sawyer, Edward Keyes | Somerville. |
| 1900 | Searles, Frank Rufus | Springfield. |
| 1901 | Simmons, Channing Chamberlain | Boston. |
| 1900 | Small, Albert Ernest | New Bedford. |
| 1901 | Snow, Henry Curtis Butler | Quincy. |
| 1900 | Spalter, Charles Marsh | New York, N. Y. |
| 1900 | Stewartson, Charlotte Dodd | Roxbury. |
| 1899 | Stowe, Irving Elmer | Brookline. |
| 1901 | Sullivan, John Joseph | South Boston. |
| 1901 | Swift, Henry Marshall | Worcester. |
| 1901 | Sylvester, Charles Porter | Dorchester. |
| 1900 | Taylor, James, Jr. | Worcester. |
| 1900 | Taylor, John Danforth | East Boston. |
| 1901 | Wales, Ernest deWolfe | Boston. |
| 1901 | Ward, Frederick Spalding | Springfield. |
| 1901 | Warren, Henry Stanley | Boston. |
| 1901 | Waterman, George Arthur | Boston. |
| 1900 | Webb, Walter James | Cambridge. |
| 1900 | Whoriskey, John Joseph | Cambridge. |
| 1901 | Wormelle, Charles Burton | Brighton. |
| 1900 | Wyer, Harry Gage | Kirkood, Mo. |
| 1900 | York, Herbert Leroy | Boston. |
| 1900 | Yost, John Dixon | U. S. Army. |

Total, 135.

## *List of Deceased Fellows.*

| Admitted. | Name. | Residence. | Date of Death. | Age. |
|---|---|---|---|---|
| 1874 | *Acland, Sir Henry Wentworth Dyke | Oxford, Eng. | Oct. 16, 1900 | 85 |
| 1876 | Bartlett, Benjamin Webber | Rowley | Aug. 6, 1900 | 50 |
| 1868 | Chenery, Elisha | Boston | Aug. 1, 1900 | 70 |
| 1832 | Cogswell, George | Bradford | April 21, 1901 | 93 |
| 1870 | Eveleth, Philemon | Marblehead | May 14, 1900 | 54 |
| 1899 | Forrest, Robert Francis | Cambridge | Sept. 18, 1900 | 27 |
| 1873 | Giddings, Theodore | Housatonic | Dec. 28, 1900 | 64 |
| 1878 | Gould, Lawrence Mervin | Hyde Park | Oct. 7, 1900 | 46 |
| 1884 | Greenleaf, Robert Willard | Boston | April 28, 1901 | 45 |
| 1893 | Hamilton, Erskine Erasmus | Springfield | Jan. 24, 1901 | 34 |
| 1875 | Hammond, Roland | Campello | July 8, 1900 | 58 |
| 1843 | Hayward, George | Boston | March 30, 1901 | 81 |
| 1897 | Howard, William Francis | Lawrence | June 15, 1900 | 31 |
| 1889 | Hyde, Seneca Tobias | Dorchester | Feb. 17, 1901 | 60 |
| 1886 | McDonough, Lawrence John | Lowell | March 2, 1901 | 42 |
| 1880 | O'Callaghan, Thomas Albert | Worcester | April 13, 1901 | 46 |
| 1868 | Osborne, George Sterne | Salem | May 26, 1901 | 62 |
| 1868 | Palmer, Harris Orlando | Hubbardston | Feb. 17, 1901 | 59 |
| 1875 | Riley, Thomas | Adams | March 4, 1901 | 57 |
| 1881 | Robbins, James Henry | Hingham | Aug. 21, 1900 | 61 |
| 1894 | Roberts, Henry Ambrose | Dorchester | Jan. 19, 1901 | 32 |
| 1897 | Ryan, John Francis | Roxbury | Sept. 1, 1900 | 29 |
| 1852 | Shurtleff, Augustine | Brookline | Jan. 27, 1901 | 74 |
| 1885 | Steadman, Augusta Alice | Amst'dam, N.Y. | | 50 |
| 1870 | Stevens, Charles Wistar | Charlestown | Jan. 25, 1901 | 64 |
| 1851 | Sullivan, John Langdon | Malden | Sept. 5, 1900 | 73 |
| 1868 | Sumner, Allen Melancthon | Boston | May 25, 1901 | 57 |
| 1866 | Tarbell, George Grosvenor | Boston | Dec. 28, 1900 | 59 |
| 1872 | Thayer, Frederick Lyman | Newton | March 4, 1901 | 52 |
| 1853 | Warren, Ephraim Lewis | Melrose | April 28, 1901 | 78 |

* Honorary. Total, 30.

The Treasurer's report was read.

Papers were read as follows:

Some Cases of Cancer Treated by the X-Rays.—By Dr. F. H. Williams, of Boston.

A Critical Note upon Clinical Methods of Measuring Blood Pressure:—from the Physiological Laboratory of Johns Hopkins Medical School.—By Mr. C. E. Brush, Jr., and Dr. W. H. Howell, of Baltimore, Md.

Practical Blood Examination:—from the Chemical Department of the Harvard Medical School.—By Dr. H. F. Hewes, of Boston.

Problems of Bacteriological Laboratories which have to do with Public Health:—from the Laboratory of the Boston Board of Health.—By Dr. H. W. Hill, of Boston.

THE ORIGIN OF OXALIC ACID FROM PROTEIN AND PROTEIN DERIVATIVES:—from the Laboratory of the Chemical Department of Tufts College Medical School.—By Dr. A. E. Austin, of Boston.

INTRACRANIAL PRESSURE AFTER HEAD INJURIES:—from the Physiological Laboratory of the Harvard Medical School.—By Dr. W. B. Cannon, of Boston.

STATEMENT OF ORIGINAL WORK DONE IN THE DEPARTMENT OF ANATOMY OF THE HARVARD MEDICAL SCHOOL, AND DESCRIPTION OF A MODEL OF THE ABDOMINAL VISCERA.—By Dr. Thomas Dwight, of Nahant.

The following delegates from other State Medical Societies were present:

*Maine.*—Dr. C. D. Smith.
*New Hampshire.*—Dr. J. W. Parsons.
*Vermont.*—Dr. H. M. Chisholm.
*New Jersey.*—Dr. S. A. Hetler.
*Pennsylvania.*—Drs. G. G. Harmon and J. J. Koser.

At 12.30 P.M. the Annual Discourse was delivered by Dr. GEORGE E. FRANCIS, of Worcester.

*Voted*, That the thanks of the Society be presented to the Orator for his very interesting and instructive address.

At 1.30 P.M. the Annual Dinner was served in Symphony Hall to nearly one thousand Fellows.

FRANCIS W. GOSS,
*Recording Secretary.*

## TREASURER'S REPORT.

APRIL 15, 1901.

During the past year the receipts of the Society have been as follows:—

| | | | |
|---|---|---|---|
| Balance June 15, 1900 | | | $8,955 87 |
| Assessments paid at Annual Meeting | | $890 00 | |
| To Treasurer at other times | | 500 00 | |
| To District Treasurers of | | | |
| Barnstable | $115 00 | | |
| Berkshire | 340 00 | | |
| Bristol North | 190 00 | | |
| Bristol South | 355 00 | | |
| Essex North | 500 00 | | |
| Essex South | 475 00 | | |
| Franklin | 150 00 | | |
| Hampden | 585 00 | | |
| Hampshire | 235 00 | | |
| Middlesex East | 265 00 | | |
| Middlesex North | 465 00 | | |
| Middlesex South | 1,350 00 | | |
| Norfolk | 1,475 00 | | |
| Norfolk South | 205 00 | | |
| Plymouth | 295 00 | | |
| Suffolk | 2,555 00 | | |
| Worcester | 875 00 | | |
| Worcester North | 285 00 | | |
| | $10,715 00 | 10,715 00 | |
| | | $12,105 00 | 12,105 00 |
| Sale of dinner tickets at Annual Meeting | | | 16 00 |
| Fine for non-attendance at Censors' Meeting | | | 5 00 |
| Income from investments, as follows:— | | | |
| Interest on Deposits, Mt. Vernon Nat. Bank | | 161 99 | |
| " " " Bay State Trust Co. | | 63 84 | |
| " " " Franklin Savings Bank | | 67 93 | |
| " " " Provident Institution for Savings (Cotting Fund) | | 32 76 | |
| " " " Roxbury Institution for Savings (Cotting Fund) | | 35 30 | |
| " " " Suffolk Savings Bank (Cotting Fund) | | 32 76 | |
| " " Annuity, Mass. Hosp. Life Ins. Co. (Shattuck Fund) | | 366 67 | |
| " " " " " " | | 65 22 | |
| " " " " " " | | 83 19 | |
| " " " " " " | | 301 71 | |
| " " United States Bond (Phillips Fund) | | 400 00 | |
| " " Massachusetts Bonds | | 70 00 | |
| | | $1,651 37 | 1,651 37 |
| Total Receipts | | | $22,733 24 |

The expenses have been—

| | | |
|---|---|---|
| President's Expense | | $64 90 |
| Secretary's Expense:— | | |
| Clerk hire | $30 00 | |
| Engrossing diplomas | 15 90 | |
| Printing | 140 67 | |
| Tubes for certificates | 3 10 | |
| Postage and incidentals | 33 91 | |
| | $223 58 | 223 58 |
| Librarian's Expense:— | | |
| Printing | 165 63 | |
| Delivering "Communications" | 81 02 | |
| Postage | 55 00 | |
| | $301 65 | 301 65 |
| Custodian's Expense:— | | |
| Moving safe | $35 00 | |
| Plumbing | 21 43 | |
| | $56 43 | 56 43 |
| Treasurer's Expense:— | | |
| Stationery, postage and printing | $77 00 | |
| Internal Revenue Stamps | 6 08 | |
| Safe Deposit Box, Bay State Tr. Co. | 10 00 | |
| Clerks at Annual Meeting | 30 00 | |
| Carriage of accounts to meeting and return | 1 00 | |
| | $124 08 | 124 08 |
| District Treasurers' Expense | | 650 57 |
| Censors' Expense | | 471 00 |
| Supervisors' Expense | | 29 54 |
| Exchange on out-of-Massachusetts checks | | .60 |
| Salaries | | 1,400 00 |
| Use of Boston Medical Library | | 262 50 |
| Legal services | | 250 00 |
| Return of overpaid assessment | | 5 00 |
| Committee on Publications :— | | |
| Printing | $1,030 07 | |
| Plates | 60 00 | |
| | $1,090 00 | 1,090 07 |
| Shattuck Lecture :— | | |
| Lecturer | $200 00 | |
| Advertising | 1 25 | |
| | $201 25 | 201 25 |
| Cotting Lunch | | 83 25 |
| *Amount carried forward,* | | $5,214 42 |

| | | | |
|---|---|---|---|
| *Amount brought forward,* | | | $5,214 42 |
| Annual Meeting :— | | | |
| Clerk | $17 00 | | |
| Type Writer | 3 00 | | |
| Stenographer | 20 00 | | |
| Rent of Mechanics Hall | 574 50 | | |
| Erecting platforms | 75 00 | | |
| Music | 40 00 | | |
| Flowers | 50 00 | | |
| Badges | 4 23 | | |
| Soap, towels and attendance | 10 15 | | |
| Card signs | 6 25 | | |
| Police | 14 00 | | |
| Dinner Tickets | 3 50 | | |
| Stationery | 46 75 | | |
| Cold Storage | 4 20 | | |
| Caterer | 2,007 56 | | |
| Cigars | 195 50 | | |
| Excursion, Boston Elevated Ry. | 13 00 | | |
| Incidentals | 8 25 | | |
| | $3,092 89 | $3,092 89 | |
| Annual Dividend :— | | | |
| Barnstable | $39 50 | | |
| Berkshire | 86 51 | | |
| Bristol North | 56 42 | | |
| Bristol South | 107 20 | | |
| Essex North | 142 93 | | |
| Essex South | 173 03 | | |
| Franklin | 37 62 | | |
| Hampden | 174 91 | | |
| Hampshire | 75 23 | | |
| Middlesex East | 90 27 | | |
| Middlesex North | 150 46 | | |
| Middlesex South | 490 86 | | |
| Norfolk | 490 86 | | |
| Norfolk South | 69 59 | | |
| Plymouth | 84 63 | | |
| Suffolk | 867 00 | | |
| Worcester | 283 99 | | |
| Worcester North | 78 99 | | |
| | $3,500 00 | 3,500 00 | |
| | | $11,807 31 | $11,807 31 |
| Balance, April 15, 1901 | | | $10,925 93 |

This balance is deposited in the Mount Vernon National Bank and the Bay State Trust Co.

The permanent investments of the Society are as follows:—

| | |
|---|---|
| Deposit in Franklin Savings Bank | $1,074 48 |
| " " Roxbury Inst. for Savings (Cotting Fund) | 1,000 00 |
| " " Provident " " " " | 1,000 00 |
| " " Suffolk Savings Bank " " | 1,000 00 |
| United States 4 per ct. Bond (Phillips Fund) | 10,000 00 |
| Massachusetts 3½ per ct. Bonds | 2,000 00 |
| Annuity Policy, Mass. Hosp. Life Ins. Co. (Shattuck Fund) | 9,166 87 |
| Three Annuity Policies, Mass. Hosp. Life Ins. Co. | 11,253 30 |
| | $36,494 65 |

EDWARD M. BUCKINGHAM,
*Treasurer.*

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The undersigned, a duly appointed committee, having examined the books of the Treasurer, find the accounts properly cast and that he has in his possession the securities called for.

HERMAN F. VICKERY,
BOSTON, MAY 15, 1901. HENRY W. BROUGHTON.

## Officers of The Massachusetts Medical Society.

1901—1902.

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CHOSEN JUNE 11, 1901.

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FRANK W. DRAPER, . . . Boston, . PRESIDENT.
AUGUSTUS C. WALKER, . . Greenfield, VICE-PRESIDENT.
EDWARD M. BUCKINGHAM, Boston, . TREASURER.
CHARLES W. SWAN, . . . Brookline, COR. SECRETARY.
FRANCIS W. GOSS, . . . . Roxbury, REC. SECRETARY.
EDWIN H. BRIGHAM, . . . Brookline, LIBRARIAN.

### Standing Committees.

*Of Arrangements.*

S. CROWELL, F. G. BALCH, FARRAR COBB,
W. H. PRESCOTT, J. C. HUBBARD, A. P. PERRY.

*On Publications.*

O. F. WADSWORTH, G. B. SHATTUCK, H. L. BURRELL.

*On Membership and Finance.*

E. G. CUTLER, L. R. STONE, F. W. GOSS,
W. ELA, C. M. GREEN.

*To Procure Scientific Papers.*

A. K. STONE, C. F. WITHINGTON, W. H. POMEROY,
J. B. BLAKE, G. L. RICHARDS.

*On Ethics and Discipline.*

G. E. FRANCIS, F. C. SHATTUCK, C. G. CARLETON,
E. COWLES, J. F. A. ADAMS.

*On Medical Diplomas.*

H. E. MARION, E. N. WHITTIER, O. F. ROGERS.

*On State and National Legislation.*

F. W. DRAPER, H. P. BOWDITCH, S. D. PRESBREY,
S. W. ABBOTT, B. H. HARTWELL.

## Presidents of District Societies—Vice-Presidents (Ex-Officiis).

[Arranged according to seniority.]

| | |
|---|---|
| F. W. Goss, | J. C. Fraser, |
| T. M. Rotch, | H. H. Lyons, |
| L. S. Brooks, | M. L. Woodruff, |
| C. E. Chase, | C. S. Holden, |
| W. H. Lathrop, | J. A. Houston, |
| C. H. Cook, | E. R. Wheeler, |
| F. B. Flanders, | H. W. Newhall, |
| M. H. Leonard, | L. Edmonds, |
| A. A. MacKeen, | H. G. Stetson. |

## Councillors.

Barnstable.—C. E. Harris, Hyannis; S. F. Haskins, Cotuit.

Berkshire.—J. F. A. Adams, Pittsfield; O. J. Brown, H. Bushnell, North Adams; W. W. Leavitt, F. K. Paddock, Pittsfield.

Bristol North.—C. A. Atwood, S. D. Presbrey, T. J. Robinson, Taunton.

Bristol South.—W. A. Dolan, J. H. Gifford, J. H. Jackson, Fall River; G. deN. Hough, C. A. Pratt, J. F. Weeks, New Bedford.

Essex North.—J. Q. Adams, Amesbury; C. G. Carleton, Lawrence; C. C. Day, Newburyport; J. F. Croston, C. E. Durant, Haverhill; J. G. McAllister, Lawrence; A. H. Pierce, West Medway; J. F. Young, Newburyport.

Essex South.—C. A. Ahearne, Lynn; F. S. Atwood, C. A. Carlton, Salem; W. W. Eaton, A. H. Harrington, Danvers; H. K. Foster, Peabody; C. W. Haddock, Beverly; A. F. Hunt, Swampscott; W. B. Little, C. F. Meader, Lynn; P. Mooney, Gloucester; G. A. Stickney, Beverly.

Franklin.—G. P. Twitchell, A. C. Walker, *Vice-President*, Greenfield.

Hampden.—W. W. Broga, D. J. Brown, Springfield; D. F. Donoghue, J. C. Hubbard, Holyoke; J. W. Hastings, Feeding Hills; C. W. Jackson, Monson; D. E. Keefe, J. E. Marsh, L. A.

Prefontaine, Springfield; J. P. Schneider, Palmer; G. L. Taylor, Holyoke.

HAMPSHIRE.—C. F. Branch, Amherst; C. A. Byrne, Hatfield; A. C. Cobb, Southampton; G. D. Thayer, Northampton.

MIDDLESEX EAST.—J. P. Bixby, North Woburn; C. W. Harlow, Melrose Highlands; F. E. Park, Stoneham; S. H. Parks, Reading.

MIDDLESEX NORTH.—R. E. Bell, W. G. Eaton, J. B. Field, W. A. Johnson, J. E. Lamoureux, T. G. McGannon, J. F. Sullivan, Lowell; G. A. Harlow, Tyngsboro'.

MIDDLESEX SOUTH.—Z. B. Adams, Framingham; D. E. Baker, Newtonville; R. E. Brown, Everett; J. E. Cleaves, Medford; C. H. Cook, Natick; E. Cowles, Waverley; J. G. Dearborn, Charlestown; A. G. Griffin, Malden; E. A. Hobbs, South Framingham; E. G. Hoitt, Marlboro'; E. D. Hooker, Arlington; R. P. Loring, Newton Centre; H. B. McIntire, Cambridge; H. P. Makechnie, West Somerville; H. E. Marion, Brighton; J. T. G. Nichols, A. L. Norris, Cambridge; G. A. Oviatt, South Sudbury; W. A. Phipps, Hopkinton; C. E. Prior, Malden; C. D. Sawin, Charlestown; F. M. Sherman, L. R. Stone, Newton; E. H. Stevens, H. P. Walcott, A. C. Webber, M. Wyman, Cambridge; J. W. Willis, H. A. Wood, Waltham.

NORFOLK.—R. F. Abbe, Dorchester; H. D. Arnold, Roxbury; E. E. Bancroft, Wellesley; F. A. Bragg, Foxboro'; J. E. Butler, Roxbury; S. Crowell, Dorchester; H. M. Cutts, F. P. Denny, Brookline; F. C. Dunbar, R. E. Edes, F. P. Emerson, G. H. French, Roxbury; I. S. Fogg, Norwood; D. H. Fuller, Jamaica Plain; I. H. Hazelton, Wellesley Hills; H. R. Hitchcock, Hyde Park; A. D. Hodges, Roxbury; G. H. Ingalls, Jamaica Plain; G. W. Kaan, Brookline; E. B. Lane, Dorchester; E. N. Libby, E. G. Morse, Roxbury; A. Mitchell, Medfield; R. M. Merrick, W. Noyes, H. V. Reynolds, Dorchester; L. F. Vickery, Jamaica Plain; A. G. Williams, Brookline.

NORFOLK SOUTH.—C. S. Adams, Wollaston; N. S. Hunting, Quincy; E. N. Mayberry, V. M. Tirrell, South Weymouth.

PLYMOUTH.—H. W. Dudley, Abington; A. E. Paine, F. J. Ripley, Brockton; F. G. Wheatley, North Abington.

SUFFOLK.—J. B. Ayer, E. H. Bradford, E. M. Buckingham, *Treasurer*, H. L. Burrell, J. F. Bush, A. T. Cabot, B. F. Campbell, D. W. Cheever, A. Coolidge, Jr., E. G. Cutler, H. Derby, F. W. Draper, *President*, T. W. Fisher, R. H. Fitz, C. F. Folsom, W. W. Gannett, Boston; M. F. Gavin, South Boston; C. M. Green, J. Homans, B. J. Jeffries, G. F. Jelly, F. I. Knight, A. L. Mason, J. H. McCollom, A. B. Morong, F. G. Morrill, A. H. Nichols, C. B. Porter, A. Post, J. J. Putnam, Boston; J. M. Putnam, Chelsea; M. H. Richardson, W. L. Richardson, T. M. Rotch, G. H. M. Rowe, F. C. Shattuck, G. B. Shattuck, J. B. Swift, H. F. Vickery, O. F. Wadsworth, J. C. Warren, J. C. White, E. N. Whittier, H. Williams, G. Wolcott, Boston.

WORCESTER.—W. P. Bowers, Clinton; P. P. Comey, W. Davis, O. H. Everett, G. E. Francis, T. H. Gage, Worcester; E. B. Harvey, Westboro'; S. P. Holbrook, East Douglas; J. O. Marble, H. M. Quinby, Worcester; W. G. Reed, Southbridge; E. W. Norwood, E. R. Wheeler, Spencer; E. V. Scribner, L. Wheeler, A. Wood, Worcester.

WORCESTER NORTH.—J. R. Greenleaf, Gardner; B. H. Hartwell, Ayer; C. H. Rice, F. H. Thompson, Fitchburg.

## Censors.

BARNSTABLE.—I. O. Cummings, Brewster; G. W. Doane, Hyannis; R. H. Faunce, Sandwich; S. F. Haskins, *Supervisor*, Cotuit; H. S. Kelley, Jr., West Dennis.

BERKSHIRE.—H. Bushnell, North Adams; H. Colt, W. W. Leavitt, *Supervisor*, W. M. Mercer, Pittsfield; W. W Scofield, Dalton.

BRISTOL NORTH.—A. S. Deane, F. A. Hubbard, J. B. Murphy, S. D. Presbrey, *Supervisor*, Taunton; A. M. Round, Norton.

BRISTOL SOUTH.—S. W. Bowen, D. E. Cone, W. A. Dolan, *Supervisor*, Fall River; A. M. Pierce, J. C. Pothier, New Bedford.

ESSEX NORTH.—C. E. Durant, *Supervisor*, Haverhill; J. Pierce, Methuen; W. W. Pillsbury, Newburyport; W. J. Sullivan, Lawrence; L. J. Young, Haverhill.

ESSEX SOUTH.—F. F. Brigham, Lynn; H. K. Foster, *Supervisor*, Peabody; J. H. Knowles, Gloucester; S. F. Quimby, Salem; F. E. Stone, Lynn.

FRANKLIN.—E. G. Best, Turner's Falls; J. W. Cram, Colrain; G. R. Fessenden, Ashfield; A. C. Walker, *Supervisor*, Greenfield; N. P. Wood, Northfield.

HAMPDEN.—J. W. Hastings, *Supervisor*, Feeding Hills; F. Holyoke, Holyoke; W. M. E. Mellen, Chicopee; P. Kilroy, S. M. Wilbur, Springfield.

HAMPSHIRE.—J. G. Hayes, Williamsburg; J. B. Learned, Florence; H. B. Perry, Amherst; G. D. Thayer, *Supervisor*, W. M. Trow, Northampton.

MIDDLESEX EAST.—D. T. Buzzell, Wilmington; M. A. Cummings, Winchester; C. W. Harlow, *Supervisor*, Melrose; A. C. Lane, Woburn; G. N. P. Mead, Winchester.

MIDDLESEX NORTH.—J. B. Field, *Supervisor*, T. F. Harrington, S. J. Johnson, W. P. Lawler, C. E. Simpson, Lowell.

MIDDLESEX SOUTH.—O. W. Collins, South Framingham; F. G. Curtis, West Newton; H. B. McIntire, *Supervisor*, Cambridge; C. D. Sawin, Charlestown; J. Tolman, Arlington.

NORFOLK.—H. M. Cutts, *Supervisor*, Brookline; C. G. Dewey, Dorchester; G. H. Francis, Brookline; H. R. Hitchcock, Hyde Park; G. B. Stevens, Roxbury.

NORFOLK SOUTH.—W. A. Drake, North Weymouth; F. C. Granger, Randolph; J. A. Gordon, J. F. Welch, Quincy; E. N. Mayberry, *Supervisor*, South Weymouth.

PLYMOUTH.—J. E. Bacon, H. F. Borden, W. P. Chisholm, Brockton; C. A. Drew, Bridgewater; H. W. Dudley, *Supervisor*, Abington.

SUFFOLK.—A. Coolidge, Jr., *Supervisor*, H. Jackson, H. A. Lothrop, J. C. Munro, G. H. Washburn, Boston.

WORCESTER.—O. H. Everett, M. F. Fallon, H. Gage, E. V. Scribner, *Supervisor*, Worcester, C. L. French, Clinton.

WORCESTER NORTH.—J. G. Henry, Winchendon; A. P. Mason, Fitchburg; E. A. Sawyer, Gardner; F. H. Thompson, *Supervisor*, Fitchburg; G. B. Underwood, West Gardner.

## Commissioners of Trials.

| | | |
|---|---|---|
| Barnstable | C. W. Milliken | Barnstable. |
| Berkshire | A. T. Wakefield | Sheffield. |
| Bristol North | F. A. Hubbard | Taunton. |
| Bristol South | H. S. Swan | Middleboro'. |
| Essex North | F. A. Howe | Newburyport. |
| Essex South | J. Allen | Topsfield. |
| Franklin | C. Bowker | Bernardston. |
| Hampden | H. W. VanAllen | Springfield. |
| Hampshire | A. G. Blodgett | Ware. |
| Middlesex East | J. S. Clark | Melrose. |
| Middlesex North | B. R. Benner | Lowell. |
| Middlesex South | A. Worcester | Waltham. |
| Norfolk | M. V. Pierce | Milton. |
| Norfolk South | N. S. Hunting | Quincy. |
| Plymouth | G. Osgood | Rockland. |
| Suffolk | F. W. Draper | Boston. |
| Worcester | C. H. Perry | Worcester. |
| Worcester North | A. W. Sidney | Fitchburg. |

## Officers of the District Medical Societies.

Barnstable.—Louis Edmonds, Harwich, *President;* E. M. Parker, South Yarmouth, *Vice-President;* J. H. Higgins, Marston's Mills, *Secretary;* G. N. Munsell, Harwich, *Treasurer.*

Berkshire.—M. L. Woodruff, Pittsfield, *President;* S. C. Burton, Pittsfield, *Vice-President;* L. C. Swift, Pittsfield, *Secretary;* W. L. Paddock, Pittsfield, *Treasurer;* W. W. Leavitt, Pittsfield, *Librarian.*

Bristol North.—C. S. Holden, Attleboro', *President;* W. Y. Fox, Taunton, *Vice-President;* R. D. Dean, Taunton, *Secretary;* W. Y. Fox, Taunton, *Treasurer;* H. B. Baker, Dighton, *Librarian.*

Bristol South.—M. H. Leonard, New Bedford, *President;* J. W. Coughlin, Fall River, *Vice-President;* A. J. Abbe, Fall River, *Secretary and Treasurer.*

Essex North.—F. B. Flanders, Lawrence, *President;* J. F. Croston, Haverhill, *Vice-President;* M. D. Clarke, Haverhill, *Secretary and Treasurer.*

ESSEX SOUTH.—H. W. Newhall, Lynn, *President;* H. J. Hall, Marblehead, *Vice-President;* C. H. Bangs, Lynn, *Secretary;* G. Z. Goodell, Salem, *Treasurer;* G. C. Littlefield, Salem, *Librarian.*

FRANKLIN.—H. G. Stetson, Greenfield, *President;* F. J. Canedy, Shelburne Falls, *Vice-President;* B. P. Croft, Greenfield, *Secretary and Treasurer.*

HAMPDEN.—L. S. Brooks, Springfield, *President;* S. A. Mahoney, Holyoke, *Vice-President;* H. C. Martin, Springfield, *Secretary and Treasurer.*

HAMPSHIRE.—J. A. Houston, Northampton, *President;* C. R. Gardner, Northampton, *Vice-President;* A. G. Minshall, Northampton, *Secretary;* A. H. Hoadley, Northampton, *Treasurer;* S. A. Clark, Northampton, *Librarian.*

MIDDLESEX EAST.—C. E. Chase, Woburn, *President;* J. W. Heath, Wakefield, *Vice-President;* E. S. Jack, Melrose, *Secretary;* C. Dutton, Wakefield, *Treasurer.*

MIDDLESEX NORTH.—W. H. Lathrop, Lowell, *President;* J. A. Gage, Lowell, *Vice-President;* W. G. Eaton, Lowell, *Secretary;* J. V. Meigs, Lowell, *Treasurer;* D. N. Patterson, Lowell, *Librarian.*

MIDDLESEX SOUTH.—C. H. Cook, Natick, *President;* E. G. Hoitt, Marlboro', *Vice-President;* A. August, Cambridge, *Secretary;* W. Ela, Cambridge, *Treasurer;* E. S. Dow, Allston, *Librarian.*

NORFOLK.—F. W. Goss, Roxbury, *President;* C. F. Withington, Roxbury, *Vice-President;* J. C. D. Pigeon, Roxbury, *Secretary and Librarian;* E. G. Morse, Roxbury, *Treasurer.*

NORFOLK SOUTH.—J. C. Fraser, East Weymouth, *President;* W. A. Drake, North Weymouth, *Vice-President;* S. W. Ellsworth, Quincy, *Secretary, Treasurer and Librarian.*

PLYMOUTH.—A. A. MacKeen, Whitman, *President;* J. H. Averill, Campello, *Vice-President;* F. H. Burnett, Brockton, *Secretary and Treasurer;* C. E. Lovell, Whitman, *Librarian.*

SUFFOLK.—T. M. Rotch, Boston, *President;* A. T. Cabot, Boston, *Vice-President;* F. J. Cotton, Boston, *Secretary;* W. H. Prescott, Boston, *Treasurer;* B. J. Jeffries, Boston, *Librarian.*

WORCESTER.—E. R. Wheeler, Spencer, *President;* S. B. Woodward, Worcester, *Vice-President;* L. C. Miller, Worcester, *Secretary;* G. O. Ward, Worcester, *Treasurer;* A. C. Getchell, Worcester, *Librarian.*

WORCESTER NORTH.—H. H. Lyons, Fitchburg, *President;* C. E. Bigelow, Leominster, *Vice-President;* W. F. Sawyer, Fitchburg, *Secretary;* E. L. Fiske, Fitchburg, *Treasurer;* A. P. Mason, Fitchburg, *Librarian.*

## SUPPLEMENT NO. I. TO CATALOGUE OF 1899.

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*Admissions reported since January 1, 1899.*

| | | |
|---|---|---|
| 1899 | Adams, William Carlton | East Taunton. |
| 1899 | Allen, Clarence Jean | Winchester. |
| 1899 | Bacon, Theodore Spaulding | Natick. |
| 1899 | Badger, George Sherwin Clarke | Brookline. |
| 1899 | Barrell, Mary Elizabeth | Worcester. |
| 1899 | Bartlett, Charles Watson | Marshfield. |
| 1899 | Bartlett, Samuel Danforth | Woburn. |
| 1899 | Brown, Daniel Rollins | Salem. |
| 1899 | Butler, Charles Shorey | Boston. |
| 1899 | Chamberlin, Theodore | Concord. |
| 1899 | Churchill, Donald | Jamaica Plain. |
| 1899 | Collins, William James | Northampton. |
| 1899 | Crandon, Le Roi Goddard | Boston. |
| 1899 | Cronin, Michael John | Boston. |
| 1899 | Cross, William Patrick | South Boston. |
| 1899 | Cummin, John White | Boston. |
| 1899 | Curtis, William Goodwin | Wollaston. |
| 1899 | Davis, Winifred | Charlestown. |
| 1899 | Donoghue, James Crowley | Boston. |
| 1899 | Dow, David Crooker | Cambridge. |
| 1899 | Downey, William Henry | Peabody. |
| 1899 | Drake, Arthur Knowlton | Tewksbury. |
| 1899 | Ellis, William Raymond | Allston. |
| 1899 | Ellsworth, Samuel Walker | Quincy. |
| 1899 | Emerson, Ernest Benjamin | Tewksbury. |
| 1899 | Everett, Eugene Ellsworth | Boston. |
| 1899 | Fair, John Francis | Boston. |
| 1899 | Fairbanks, Arthur Willard | Boston. |
| 1899 | Field, Harvey Adams | Dorchester. |
| 1899 | Forrest, Robert Francis | Cambridge. |
| 1898 | Fullerton, Walter Wilson | Brockton. |
| 1899 | Gallagher, William Howard | Malden. |

| | | |
|---|---|---|
| 1899 | Garland, George Franklin | Waltham. |
| 1899 | Gavin, John Harrison | Roxbury. |
| 1899 | Grady, Patrick Anthony Salmon | Clinton. |
| 1899 | Grimes, James Henry | Boston. |
| 1899 | Hamilton, Annie Lee | Boston. |
| 1899 | Haviland, Nathaniel Clark Bacon | Holliston. |
| 1899 | Hayes, Frederick Legro | Roxbury. |
| 1899 | Healy, Thomas Raymond | Newburyport. |
| 1899 | Hinchey, Richard | Roxbury. |
| 1899 | Hinckley, James William | Boston. |
| 1899 | Howe, Joseph Dimock | Becket. |
| 1899 | Hyde, Frederick Tanquary | Waltham. |
| 1899 | Jackson, Alexander Washington | Dorchester. |
| 1899 | Jenness, Burt Franklin | Tewksbury. |
| 1899 | Knight, Howard Webster | Dorchester. |
| 1899 | Lecompte, Walter Augustus | Newton Centre. |
| 1899 | Libby, Mary Gordon | Lowell. |
| 1899 | McAllister, Frederick Danforth | Worcester. |
| 1899 | Macdonald, Donald William | Brookline. |
| 1899 | Mackie, William Charles | Worcester. |
| 1899 | Mansur, Leon Wallace | Boston. |
| 1899 | May, John Shepard | Roxbury. |
| 1899 | Merrill, Quincy Heald | Fitchburg. |
| 1899 | O'Keefe, Daniel Thomas | Jamaica Plain. |
| 1899 | O'Sullivan, John Joseph | Lawrence. |
| 1899 | Overlock, Melvin George | Worcester. |
| 1899 | Packard, George Henry | Medford. |
| 1899 | Page, Frederick Carroll | Brookline. |
| 1899 | Parker, Ralph Walker | Lowell. |
| 1899 | Parker, Walter Henry | Dorchester. |
| 1899 | Pease, Charles Wood | Tewksbury. |
| 1899 | Peirce, George Alphonso | Tewksbury. |
| 1899 | Reagh, Arthur Lincoln | West Roxbury. |
| 1899 | Reid, Eustace Palmer | Boston. |
| 1899 | Shurtleff, Walter Davis | Carver. |
| 1899 | Strong, Frederick Finch | Boston. |
| 1899 | Sughrue, Dennis Francis | Roxbury. |
| 1899 | Tenney, Elmer Seth | Roxbury. |
| 1899 | Thompson, Richard Henry | Malden. |
| 1899 | Thorn, Edwin Cyrus | Deerfield. |
| 1899 | Walker, Alexander Taylor | Falmouth. |
| 1899 | Walsh, Charles Joseph | Cambridge. |
| 1899 | Watson, Frank Gilman | Roxbury. |
| 1899 | White, Michael William | Boston. |
| 1899 | Wood, Mary Anna | Pittsfield. |
| 1899 | Woodbury, Frank Taylor | Wakefield. |
| 1899 | Young, Emma Elizabeth | Chelsea. |

## *List of Deceased Fellows.*

| Admitted. | Name. | Residence. | Date of Death. | Age. |
|---|---|---|---|---|
| 1866 | Bigelow, Orvis Furman | Amherst | Feb. 2, 1899 | 63 |
| 1875 | Bowen, Seranus | Roxbury | April 7, 1899 | 59 |
| 1866 | Brigham, Frank Whiting | Shrewsbury | Feb. 28, 1899 | 57 |
| 1869 | Clough, Benjamin Franklin | Worcester | May 8, 1899 | 61 |
| 1858 | Godding, William Whitney | Washington, D.C | May 6, 1899 | 68 |
| 1870 | Hurd, Edward Payson | Newburyport | Feb. 24, 1899 | 60 |
| 1856 | Lovejoy, Oliver Sherwin | Haverhill | June 26, 1898 | 72 |
| 1894 | McMillan, Wallace | Dorchester | March 31, 1899 | 31 |
| 1896 | Manson, Frank George | Billerica | Oct. 19, 1898 | 32 |
| 1845 | Minot, Francis | Boston | May 11, 1899 | 78 |
| 1867 | Quincy, Henry Parker | Dedham | March 11, 1899 | 60 |
| 1897 | Ronayne, David Patrick | Boston | April 2, 1899 | 32 |
| 1848 | Sparrow, William Edward | Mattapoisett | May 15, 1899 | 75 |

The following have been allowed to retire:

William G. Allen, of Mansfield.
David Choate, of Salem.
Frederick H. Hooper, of New Bedford.
Edwin A. Kemp, of Danvers.
Samuel J. McDougall, of Boston.
John H. Richardson, of Medfield.
Alexander D. Sinclair, of Boston.
Emerson Warner, of Worcester.
Orin Warren, of West Newbury.

The following have been allowed to resign:

William C. Billings, of New York, N. Y.
Joseph A. Capps, of Chicago, Ill.
Dwight E. Cone, of Binghamton, N. Y.
Charles F. Foye, of Haverhill.
Fred B. Jewett, of Howard, R. I.
Nathaniel H. Kirby, of Denver, Col.
Erastus E. Maryott, of Millbrook, N. Y.
John P. Moore, of Long Island City, N. Y.
John W. Perkins, of Kansas City, Mo.
Herbert L. Smith, of Nashua, N. H.
William S. Thayer, of Baltimore, Md.
Jerome H. Waterman, of New York, N. Y.

The following, having neglected or declined to pay their assessment dues for the past three years, have been deprived of the privileges of membership, in accordance with By-Law VIII.:

Oliver G. Burgess, of Boston.
William J. Chisholm, of Springfield.

Edmond F. Cody, of New Bedford.
Mary P. Eddy, of Beirut, Syria.
Horace S. Everett, of Boston.
Oscar H. Holder, of New York, N. Y.
George W. Huse.
Herbert E. Mace, of Boston.
Timothy E. McOwen, of Lowell.
William E. McPherson, of Charlestown.
Joseph F. Mulcahy, of Lowell.
John L. Ryan, of Malden.
Benjamin A. Sawyer, of Haverhill.
Ella G. Stone, of Lawrence.
Frederick Tigh, of Newburyport.
Hilbert B. Tingley.
Benjamin N. Towle, of Charlestown.
George F. Webber, of Fairfield, Me.
Sydney Y. Wynne, of Redlands, Cal.

## SUPPLEMENT No. II. TO CATALOGUE OF 1899.

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*Admissions reported since June 13, 1899.*

| | | |
|---|---|---|
| 1900 | Abbott, Edson Moses | Woburn. |
| 1900 | Alfred, James | Brockton. |
| 1899 | Andrews, Harold Virgil | Boston. |
| 1899 | Atkinson, Roger Trowbridge | Boston. |
| 1899 | Bailey, William Thomas | Boston. |
| 1899 | Baker, Lewis Forrester | Fitchburg. |
| 1900 | Baketel, Harrie Sheridan | Melrose Highlands. |
| 1899 | Barnes, Harry Aldrich | Boston. |
| 1900 | Barrell, Charles Lowell | Boston. |
| 1900 | Barton, John Alfred | Fitchburg. |
| 1899 | Beckley, Chester Charles | Lancaster. |
| 1899 | Birge, Russell Hall | Cleveland, Ohio. |
| 1900 | Black, George Luke | Lowell. |
| 1899 | Bridgman, Burt Nicholas | Jamaica Plain. |
| 1899 | Briggs, Lloyd Vernon | Boston. |
| 1899 | Brown, Alphonso Bickford | Newburyport. |
| 1899 | Brown, Anthony Leopold | Springfield. |
| 1899 | Buck, Maurice Allen | Billerica. |
| 1899 | Burke, William Henry | Cambridge. |
| 1899 | Burleigh, Robert Fletcher | South Braintree. |
| 1899 | Burns, Frederick Stanford | Boston. |
| 1899 | Buttler, Charles Voorhees | Worcester. |
| 1900 | Cahill, John Thomas | Lawrence. |
| 1900 | Callahan, Joseph Thomas | Woburn. |
| 1900 | Carothers, William Harsha | Melrose. |
| 1899 | Carroll, John Philip | Woburn. |
| 1900 | Cate, Frederick Symon | Melrose. |
| 1899 | Chapman, Charles Ratchford | Springfield. |
| 1900 | Charteris, Mary Alena | Worcester. |
| 1900 | Chase, Edwin Llewellyn | Shrewsbury. |
| 1900 | Choate, Horace Henry | Salem. |
| 1900 | Clap, Edmund Wright | Boston. |
| 1900 | Cone, Dwight Eleazer | Fall River. |
| 1900 | Crane, Carl Custer | Melrose. |
| 1899 | Crawford, Francis Xavier | South Boston. |
| 1900 | Crittenden, Samuel Wright | Mattapan. |
| 1900 | Cronin, Jeremiah Augustine | South Boston. |
| 1899 | Crowley, Ambrose | Cambridge. |
| 1900 | Culbert, Robert Ronayne | Roxbury. |

| | | |
|---|---|---|
| 1900 | Cummings, Frederic Russell | Boston. |
| 1900 | Currier, William Eugene | Boston. |
| 1899 | Dalton, Martin James | Melrose. |
| 1900 | Daudelin, Simeon Alphonse | Worcester. |
| 1899 | Davis, Charles Henry | Beverly. |
| 1899 | Davis, Lincoln | Boston. |
| 1900 | Dennett, Alonzo Gustin | Lowell. |
| 1900 | Dodge, Arthur Malcolm | Boston. |
| 1899 | Dole, Charles Frederick | South Boston. |
| 1900 | Dorgan, Joseph Aloysius | Lawrence. |
| 1900 | Dorman, Albert Barnes | Winthrop. |
| 1900 | Duckering, William West | Dorchester. |
| 1900 | Easton, Elwood Tracy | Boston. |
| 1900 | Eaton, Lilley | Winchester. |
| 1900 | Elliot, Henry Libbey | Salem. |
| 1900 | Elliott, Richard Andrew | Avon. |
| 1899 | Ely, Richard Skinner | West Townsend. |
| 1900 | Emery, George Edwin | Worcester. |
| 1899 | Estabrook, Charles Taylor | Worcester. |
| 1900 | Finch, George Hardy | Springfield. |
| 1899 | Flynn, John Leo | Cambridge. |
| 1900 | French, Charles Ephraim | Brookline. |
| 1900 | Golden, Lazarus | Boston. |
| 1900 | Goodwin, Harold Carl | Tewksbury. |
| 1899 | Greenough, Clara Mary | Deerfield. |
| 1900 | Haines, Ignatius | Cambridge. |
| 1899 | Hammond, William John | Dorchester. |
| 1899 | Handy, Harry Tucker | Jamaica Plain. |
| 1900 | Hart, Henry Brown | East Dennis. |
| 1899 | Haskins, Frank Henry | Springfield. |
| 1899 | Hatch, Edward Sparhawk | Brighton. |
| 1899 | Hathaway, John Gael | New Bedford. |
| 1900 | Hazlett, Olive Binkley | South Framingham. |
| 1900 | Heald, Charles Gerry | Pepperell. |
| 1900 | Heaton, Thomas Henry | Cambridge. |
| 1900 | Hixon, Edwin Colfax | Cambridge. |
| 1900 | Holmes, Harry Bigelow | Adams. |
| 1900 | Hopkins, Bertrand Hiram | Ayer. |
| 1899 | Howard, Joseph Francis | Lawrence. |
| 1900 | Howell, Thomas | Worcester. |
| 1900 | Hurd, Albert Gordon | Millbury. |
| 1899 | Hurd, Randolph Campbell | Newburyport. |
| 1899 | Jackson, William Andrew, Jr. | Haydenville. |
| 1899 | Jaques, Louis Désiré Onésiphoré | Worcester. |
| 1900 | Kinney, William D'Arcy | Osterville. |
| 1899 | Knight, Charles Eugene | Rockland. |
| 1899 | Knowlton, John Greenleaf Whittier | Boston. |
| 1900 | Ladd, Maynard | Boston. |
| 1899 | Laighton, Florence Marion | New York, N. Y. |
| 1899 | LaMotte, Henry | Northampton. |
| 1899 | Latham, Benoni Mowry | Mansfield. |
| 1899 | Lawrence, Nellie Louise | Roxbury. |
| 1900 | Leslie, Herbert Granville | Amesbury. |

| | | |
|---|---|---|
| 1900 | Livingston, Ernest George | Lowell. |
| 1899 | Lougee, William Wheeler | Malden. |
| 1900 | Lovell, Martha Eleanor | Roxbury. |
| 1899 | Lynch, Charles Francis | Springfield. |
| 1900 | McDermott, Joseph Edward | Boston. |
| 1899 | MacDonald, William Joseph | Charlestown. |
| 1900 | McEvoy, George Albert | Roxbury. |
| 1899 | McIntosh, John David | Rockland. |
| 1900 | McMurray, Francis Michael | Fitchburg. |
| 1899 | Mains, Charles Frederick | Dorchester. |
| 1900 | Masten, Charles Howard | Worcester. |
| 1899 | Merriam, Franklin Henry | Sterling. |
| 1900 | Milot, Wilfrid François | Taunton. |
| 1900 | Morin, Jacob Mauritz | Brockton. |
| 1900 | Murphy, Edward Vincent | Fall River. |
| 1899 | Nason, Arthur Clark | Newburyport. |
| 1900 | Nelson, John William | Waltham. |
| 1899 | Newton, Aaron Lewis | Northfield. |
| 1900 | Nicola, Charles Chesterfield | South Lancaster. |
| 1899 | Nielsen, Edwin Björne | West Newton. |
| 1899 | Northrop, Clarence Clark | Boston. |
| 1900 | Norton, George Paul | Fitchburg. |
| 1899 | Noyes, Henry Allen | Pittsfield. |
| 1900 | O'Brien, Thomas James | South Boston. |
| 1899 | O'Connor, John Edward | Fall River. |
| 1900 | O'Donnell, James Coughlin | Lowell. |
| 1899 | Ohnesorg, Karl | U. S. Navy. |
| 1900 | Patterson, William Francis | Charlestown. |
| 1899 | Paul, Luther Gordon | Boston. |
| 1900 | Peck, Luke Baker | Brookline. |
| 1899 | Perry, Edgar | Boston. |
| 1900 | Pillsbury, Boyden Harlin | Lowell. |
| 1899 | Place, Ralph Waldo | Boston. |
| 1900 | Prevaux, John Jacob | Haverhill. |
| 1900 | Purvis, Alice Hatheway | Roxbury. |
| 1900 | Randall, George Merrill | Lowell. |
| 1900 | Riley, Elizabeth Angela | Roxbury. |
| 1899 | Russell, Simon James | Springfield. |
| 1899 | Sayward, William Henry, Jr. | Dorchester. |
| 1900 | Schmidt, Frederick Sextus | Jamaica Plain. |
| 1900 | Schorer, Cornelia Bernhardine Johanna | Boston. |
| 1900 | Sears, Stephen Hull | Yarmouthport. |
| 1899 | Sharp, Walter Nevin | Saxonville. |
| 1900 | Smith, Frank Herbert | Hadley. |
| 1899 | Smith, Harry Atherton | West Roxbury. |
| 1899 | Snow, Frederick Stedman | Roxbury. |
| 1900 | Soutter, Robert | Boston. |
| 1899 | Spooner, Henry Garrettson | Boston. |
| 1899 | Stevens, Ralph Emerson | Marlborough. |
| 1899 | Stockwell, Edgar Washburn | New Marlborough. |
| 1899 | Stockwell, Herbert Emmons | Stockbridge. |
| 1899 | Stoddard, Mortimer James | Springfield. |
| 1900 | Stone, Warren Buxton | New Bedford. |

| | | |
|---|---|---|
| 1900 | Street, Lionel Alexander Burnet | Brookline. |
| 1900 | Swain, Howard Townsend | Boston. |
| 1900 | Symonds, Alice Gertrude | Haverhill. |
| 1899 | Thorning, William Burton | Winchendon. |
| 1899 | Tilden, Irving Niles | New Bedford. |
| 1900 | Upham, Emily Clark | Boston. |
| 1899 | von Groll, Maximilian Charles | Roxbury. |
| 1899 | Washburn, Frank Hall | Lynn. |
| 1900 | Wells, Abner Toothaker | Jamaica Plain. |
| 1900 | Wheeler, Lucia Anna | Worcester. |
| 1900 | Wilkins, George Clarence | South Boston. |
| 1899 | Williams, Hugh | Boston. |
| 1899 | Woodbury, Herbert Elwell | Natick. |
| 1899 | Woods, Charles Edwin | Lunenburg. |

## *List of Deceased Fellows.*

| Admitted. | Name. | Residence. | Date of Death. | Age. |
|---|---|---|---|---|
| 1878 | Bartlett, George Pinkham | Woburn | March 27, 1900 | 49 |
| 1873 | Brechin, William Pitt | Boston | Dec. 10, 1899 | 48 |
| 1852 | Bronson, John Richardson | Attleborough | May 9, 1900 | 70 |
| 1852 | Chamberlain, Cyrus Nathaniel | Andover | July 18, 1899 | 70 |
| 1852 | Deane, Adam Calhoun | Greenfield | Nov. 7, 1899 | 76 |
| 1885 | French, George Morrill | Malden | Feb. 23, 1900 | 45 |
| 1854 | Gilbert, John Henry | Quincy | Aug. 3, 1899 | 72 |
| 1878 | Graves, Frank Walker | Woburn | March 13, 1900 | 57 |
| 1854 | Greene, Francis Coles | Easthampton | Aug. 2, 1899 | 72 |
| 1895 | Hall, Milton Wilder | Roxbury | Jan. 9, 1900 | 51 |
| 1870 | Hastings, William Henry Howe | Boston | Feb. 16, 1900 | 60 |
| 1864 | Hutchins, Isaiah | West Acton | Aug. 7, 1899 | 69 |
| 1854 | Jenks, Thomas Leighton | Boston | Oct. 31, 1899 | 69 |
| 1883 | Kemble, Lawrence Grafton | Salem | Aug. 8, 1899 | 37 |
| 1897 | Kingsley, Charles | Marlborough | Dec. 29, 1899 | 30 |
| 1864 | McLaughlin, James Augustin | Boston | Sept. 8, 1899 | 85 |
| 1839 | Marsh, Austin | Carlisle | Feb. 2, 1900 | 88 |
| 1846 | Morse, Luther Blodgett | Watertown | May 26, 1900 | 79 |
| 1891 | O'Hearn, William Henry | Lawrence | June 4, 1900 | 30 |
| 1882 | *Paget, Sir James | London, Eng. | Dec. 30, 1899 | 85 |
| 1854 | Perkins, Edward Augustus | Boston | Feb. 20, 1900 | 72 |
| 1874 | Pierce, Gardner Carpenter | Ashland | May 18, 1900 | 62 |
| 1865 | Plunkett, Francis Charles | Lowell | Nov. 29, 1899 | 57 |
| 1869 | Stackpole, Paul Augustine | Dover, N. H. | Nov. 28, 1900 | 86 |
| 1862 | Street, Charles Carroll | Boston | May 7, 1900 | 64 |
| 1881 | Sturgis, Russell, Jr. | Boston | July 17, 1899 | 42 |
| 1851 | Warren, George Washington | West Boylston | April 22, 1900 | 81 |
| 1888 | Woodbridge, Luther Dana | Williamstown | Nov. 3, 1899 | 48 |
| 1880 | Wright, Walter Melvin | Orange | June 24, 1899 | 53 |

* Honorary.

The following name has been changed:

Sosnoski, Philip, . . to . . Sumner, Philip Sosnoski.

The following have been placed on the retired list:

Daniel A. Cleaveland, of Hyannis.
James R. Deane, of Newton Highlands.
Samuel L. Dutton, of Newton Centre.
Thomas H. Gage, of Worcester.

William Ingalls, of Roxbury.
Gustav Liebmann, of Boston.
Rollin H. Phelps, of Littleton.
Albert B. Robinson, of Roxbury.
Alonzo L. Stickney, of Ashburnham.
J. Langdon Sullivan, of Malden.
Oscar F. Swasey, of Beverly.
Peleg Wadsworth, of Malden.
John W. Willis, of Waltham.

The following have been allowed to resign:

Chauncy Adams, of Concord, N. H.
George W. Cutler, of Waltham.
Edward J. Cutter, of Leominster.
Henry A. Deane, of East Windsor, Conn.
George W. Holden, of Denver, Col.
Hairabed S Jelalian, of Boston.
James A. Keown, of Lynn.
Isaac Louis, of Boston.
Henry P. Lovewell, of Providence, R. I.
Paul Plummer, of Collinsville, Conn.
William H. Russell, of Ipswich.
Mary Sanderson, of Worcester.
George P. Sprague, of Lexington, Ky.
Eugene P. Stone, of United States Navy.
Elsie B. Taylor, of Boston.
Herbert Warren, of Worcester.

The following, having neglected or declined to pay their assessment dues for the past three years, have been deprived of the privileges of membership in accordance with By-Law VIII.:

Charles E. Adams, of Santa Clara, Cal.
Henry W. Brown, of Binghamton, N. Y.
Lewis N. Bump, of Somerville.
Edwin H. Codding.
Abraham I. Dinenson, of Boston.
William L. Faxon, of Middleborough.
George W. Gates, of San Francisco, Cal.
Charles A. Gould, of Newton Highlands.
Samuel K. Harriman, of Providence, R. I.
Patrick H. Keefe, of Providence, R. I.
Horace M. Locke, of Sturbridge.
Charles Mackin, of Milford.
Albert N. Mehrenlender, of New York, N. Y.
James W. Moore, of New York, N. Y.
Laurentius M. Nason, of Worcester.
Julius Selva, of Coainto, Nicaragua.
Harry J. Thompson, of W. Philadelphia, Pa.
James N. Tobin, of Pittsfield.
Melville E. Webb, of Boston.

*Admissions reported since June 12, 1900.*

| | | |
|---|---|---|
| 1900 | Aldrich, Albert Clinton | Somerville. |
| 1901 | Allen, Freeman | Boston. |
| 1900 | Atwood, Charles Fenner | Hyde Park. |
| 1900 | Bacon, Newton Samuel | Cambridge. |
| 1901 | Balmer, William Edward | Boston. |
| 1900 | Barnes, James Arthur | Worcester. |
| 1900 | Bedell, Charles Ellsworth | Somerville. |
| 1901 | Beebe, Theodore Chapin, Jr. | Boston. |
| 1901 | Beede, M Josephine | Boston. |
| 1901 | Bertrand, Alexis Evariste | Lowell. |
| 1901 | Bicknell, Ralph Emerson | Swampscott. |
| 1900 | Blair, George Kenniston | Salem. |
| 1901 | Blake, Le Grand | Milford. |
| 1900 | Bolster, Augustus Sargent | Worcester. |
| 1900 | Boutelle, Harry Clifton | Dorchester. |
| 1901 | Boyer, Joseph Napoleon | Springfield. |
| 1901 | Bryant, Fred | Worcester. |
| 1901 | Burke, Walter Thomas | Boston. |
| 1901 | Burnham, Melvin Page | Tewksbury. |
| 1901 | Clark, Edward James | Lowell. |
| 1901 | Clark, Ezra Warren | Brockton. |
| 1900 | Connolly, John Matthew | Dorchester. |
| 1901 | Coolidge, Sumner | Watertown. |
| 1900 | Cornwell, Herbert Cerdá deVilarrestau | Cambridge. |
| 1900 | Creesy, Everett Larcom | Beverly. |
| 1901 | Cushing, Adelaide Olga | Roxbury. |
| 1899 | Davis, Percy Guy | Deerfield. |
| 1901 | DeGrandpré, Louis Philippe | Worcester. |
| 1901 | Denning, William Edward | Worcester. |
| 1901 | Donlan, Charles Edwin | Lowell. |
| 1901 | Doucett, Frederick Luke | East Weymouth. |
| 1901 | Dresser, Arthur James | Tewksbury. |
| 1901 | Drummond, Edward Augustine | Providence, R. I. |
| 1901 | Eastman, Alexander Crane | Boston. |
| 1900 | Elmere, John Alfred | Boston. |
| 1900 | Emerson, William Robie Patten | Boston. |
| 1900 | Ewing, George Winburn | Middleton. |
| 1900 | Ferguson, Edward Hugh | Dorchester. |
| 1901 | Ferguson, Robert Henry | Dorchester. |
| 1901 | Forster, Robert William | Lawrence |
| 1901 | Fraser, Margaret Ethel Victoria | Boston. |
| 1901 | Friedman, Leo Victor | Boston. |
| 1901 | Galvin, William | Blackinton. |
| 1900 | Gardner, Harrie Milton | Athol. |
| 1900 | Gay, William Frederick | Boston. |
| 1901 | Germain, Harry Hosmer | Boston. |
| 1901 | Goodrich, Harriet Rawson | Salem. |
| 1901 | Gray, Alice Maud | Dorchester. |
| 1901 | Greene, Daniel Crosby, Jr. | Boston. |
| 1901 | Hale, Willis Le Baron | Attleboro' Falls. |
| 1901 | Hall, James Frank | Lowell. |
| 1900 | Hall, Rufus Hacker | Everett. |
| 1900 | Harding, Walter Allen | Everett. |
| 1901 | Harriman, Charles Henry | Whitinsville. |

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|---|---|---|
| 1900 | Harris, William DeBlois | Lynn. |
| 1900 | Hawes, Alfred Taylor | Lynn. |
| 1901 | Hazen, Robert | Boston. |
| 1901 | Healy, Daniel Laurence | South Framingham. |
| 1901 | Hill, George Sumner | Brookline. |
| 1900 | Hill, Hibbert Winslow | Jamaica Plain. |
| 1900 | Holt, Frank Hammett | Boston. |
| 1901 | Howard, Frederick Hollis | Williamstown. |
| 1900 | Howell, William Westcott | West Roxbury. |
| 1901 | Hubbard, George William | South Hadley. |
| 1901 | Jack, Lewis Harlow | Boston. |
| 1901 | Johnson, David Joseph | Roxbury. |
| 1901 | Johnson, Walter Sydney | Boston. |
| 1901 | Jouett, Fred Robert | Cambridge. |
| 1900 | Kepler, Charles Ober | Boston. |
| 1901 | King, James Aldrich | Lynn. |
| 1900 | Kingsbury, Charles Henry | Oxford. |
| 1901 | Lambert, Fred DeForest | Salem. |
| 1900 | Lane, Walter Appleton | Milton. |
| 1901 | Lawrence, Arthur Abbott | Milton. |
| 1900 | Legg, Arthur Thornton | Chelsea. |
| 1901 | Lewis, George Fred | New Bedford. |
| 1900 | McCormick, Alfred Hugh | Northampton. |
| 1900 | McGee, Fannie Maria | Waban. |
| 1900 | McGirr, Felix Francis | Cambridge. |
| 1901 | McLeod, John Scott | Roxbury. |
| 1900 | McNair, Robert Hamilton | Longmeadow. |
| 1900 | McNiff, William Joseph | East Pepperell. |
| 1901 | Mandell, Augustus Hamlin | New Bedford. |
| 1901 | Marcy, Henry Orlando, Jr. | Boston. |
| 1900 | Merritt, Victor Sulviro | Haydenville. |
| 1900 | Miller, Jared Homer | North Brookfield. |
| 1900 | Miller, Samuel Osgood | Three Rivers. |
| 1901 | Mitchell, William | Highlandville. |
| 1900 | Murphy, Charles Oscar | Roxbury. |
| 1900 | Murray, Charles Albert | Cambridge. |
| 1901 | Newton, LeRoy Allan | Greenfield. |
| 1901 | Nicola, Mary Byington | South Lancaster. |
| 1900 | Nightingale, James | Worcester. |
| 1901 | O'Connell, Delia Maria | Roxbury. |
| 1901 | Odiorne, Walter Burlingame | Boston. |
| 1900 | Orr, Samuel Sanford | Weston. |
| 1901 | Osgood, Robert Bayley | Boston. |
| 1900 | Peggs, Harry MacDonald | Somerville. |
| 1901 | Peterson, John Adna | Hingham. |
| 1900 | Pettigrew, Richard Richardson | Roslindale. |
| 1901 | Pike, Winfred Carle | Dorchester. |
| 1901 | Pitta, João Carlos daSilva | New Bedford. |
| 1900 | Pomeroy, Harris Starr | Peabody. |
| 1901 | Potter, Alexander Carleton | Boston. |
| 1900 | Provandie, Paul Hector | Melrose Highlands |
| 1900 | Reilly, James Aloysius | South Boston. |
| 1900 | Rich, Edwin Willis | Cambridge. |
| 1901 | Rosa, Henry Antonio | Fall River. |
| 1900 | Rust, Frank Lee Drummond | Boston. |
| 1901 | Ryder, Charles Edward | South Boston. |
| 1901 | Ryder, George Hale | Boston. |
| 1901 | Saunders, Joseph Henry | Brookline. |
| 1901 | Sawyer, Edward Keyes | Somerville. |
| 1900 | Searles, Frank Rufus | Springfield. |
| 1901 | Simmons, Channing Chamberlain | Boston. |
| 1900 | Small, Albert Ernest | New Bedford. |

| | | |
|---|---|---|
| 1901 | Snow, Henry Curtis Butler | Quincy. |
| 1900 | Spalter, Charles Marsh | New York, N. Y. |
| 1900 | Stewartson, Charlotte Dodd | Roxbury. |
| 1899 | Stowe, Irving Elmer | Brookline. |
| 1901 | Sullivan, John Joseph | South Boston. |
| 1901 | Swift, Henry Marshall | Worcester. |
| 1901 | Sylvester, Charles Porter | Dorchester. |
| 1900 | Taylor, James, Jr. | Worcester. |
| 1900 | Taylor, John Danforth | East Boston. |
| 1901 | Wales, Ernest de Wolfe | Boston. |
| 1901 | Ward, Frederick Spalding | Springfield. |
| 1901 | Warren, Henry Stanley | Boston. |
| 1901 | Waterman, George Arthur | Boston. |
| 1900 | Webb, Walter James | Cambridge. |
| 1900 | Whoriskey, John Joseph | Cambridge. |
| 1901 | Wormelle, Charles Burton | Brighton. |
| 1900 | Wyer, Harry Gage | Kirkood, Mo. |
| 1900 | York, Herbert Leroy | Boston. |
| 1900 | Yost, John Dixon | U. S. Army. |

## *List of Deceased Fellows.*

| Admitted. | Name. | Residence. | Date of Death. | Age. |
|---|---|---|---|---|
| 1874 | *Acland, Sir Henry Wentworth Dyke | Oxford, Eng. | Oct. 16, 1900 | 85 |
| 1876 | Bartlett, Benjamin Webber | Rowley | Aug. 6, 1900 | 50 |
| 1868 | Chenery, Elisha | Boston | Aug. 1, 1900 | 70 |
| 1832 | Cogswell, George | Bradford | April 21, 1901 | 93 |
| 1870 | Eveleth, Philemon | Marblehead | May 14, 1900 | 54 |
| 1899 | Forrest, Robert Francis | Cambridge | Sept. 18, 1900 | 27 |
| 1873 | Giddings, Theodore | Housatonic | Dec. 28, 1900 | 64 |
| 1878 | Gould, Lawrence Mervin | Hyde Park | Oct. 7, 1900 | 46 |
| 1884 | Greenleaf, Robert Willard | Boston | April 28, 1901 | 45 |
| 1893 | Hamilton, Erskine Erasmus | Springfield | Jan. 24, 1901 | 34 |
| 1875 | Hammond, Roland | Campello | July 8, 1900 | 58 |
| 1843 | Hayward, George | Boston | March 30, 1901 | 81 |
| 1897 | Howard, William Francis | Lawrence | June 15, 1900 | 31 |
| 1889 | Hyde, Seneca Tobias | Dorchester | Feb. 17, 1901 | 60 |
| 1886 | McDonough, Lawrence John | Lowell | March 2, 1901 | 42 |
| 1880 | O'Callaghan, Thomas Albert | Worcester | April 13, 1901 | 46 |
| 1868 | Osborne, George Sterne | Salem | May 26, 1901 | 62 |
| 1868 | Palmer, Harris Orlando | Hubbardston | Feb. 17, 1901 | 59 |
| 1875 | Riley, Thomas | Adams | March 4, 1901 | 57 |
| 1881 | Robbins, James Henry | Hingham | Aug. 21, 1900 | 61 |
| 1894 | Roberts, Henry Ambrose | Dorchester | Jan. 19, 1901 | 32 |
| 1897 | Ryan, John Francis | Roxbury | Sept. 1, 1900 | 29 |
| 1852 | Shurtleff, Augustine | Brookline | Jan. 27, 1901 | 74 |
| 1885 | Steadman, Augusta Alice | Amst'dam, N.Y. | Feb. 1, 1901 | 50 |
| 1870 | Stevens, Charles Wistar | Charlestown | Jan. 25, 1901 | 64 |
| 1851 | Sullivan, John Langdon | Malden | Sept. 5, 1900 | 73 |
| 1868 | Sumner, Allen Melancthon | Boston | May 25, 1901 | 57 |
| 1866 | Tarbell, George Grosvenor | Boston | Dec. 28, 1900 | 59 |
| 1872 | Thayer, Frederick Lyman | Newton | March 4, 1901 | 52 |
| 1853 | Warren, Ephraim Lewis | Melrose | April 28, 1901 | 78 |

* Honorary.

The following have been restored to the privileges of membership:

| | |
|---|---|
| Fourtin, Edmund Randolph Peaslee | Waltham. |
| Grover, George Wheelock | Sheffield. |
| Lyons, Christopher Philip | Boston. |

The following names have been changed:

Frost, Flora Eva, to Moody, Flora Eva.
Kelly, Jane Downes, to Sabine, Jane Downes.
Lougee, Mary Williamson, to Johnson, Mary Williamson.
Macarthy, George Elden, to MacArthur, George Elden.

The following have been placed on the retired list:

Robert F. Andrews, of Gardner.
John J. Colton, of Lowell.
Susan E. Crocker of Boston.
Thomas H. Dearing, of Braintree.
Samuel A. Green, of Boston.
George N. Munsell, of Harwich.
Charles E. Vaughan, of Santa Barbara, Cal.
Augustus C. Walker, of Greenfield.

The following have been allowed to resign:

Lyman Allen, of Burlington, Vt.
John E. Bradbury, of Denver, Col.
Wencelas J. B. Chagnon, of St. Hyacinthe, P. Q.
George W. Chamberlain, of Springfield.
Edward M. Child, of Brooklyn, N. Y.
Alice L. Cobb, of Florence, Italy.
Frank A. Dunbar, of Bonita, Cal.
Luther H. Gulick, of Brooklyn, N. Y.
Augustus S. Knight, of New York, N. Y.
John D. McIntosh, of Rockland.
John E. McKenty, of New York, N. Y.
Frederick C. Page, of Woodstock, Ill.
Richard M. Pearce, of Philadelphia, Pa.
Katherine F. Peckham, of Putnam Heights, Conn.
John E. Pratt, of Dumont, N. J.
Will H. Swan, of Colorado Springs, Col.
George H. Thomas, of Kansas City, Mo.
Gertrude W. VanPelt, of Point Loma, Cal.

The following, having neglected or declined to pay their assessment dues for the past three years, have been deprived of the privileges of membership, in accordance with By-Law VIII.:

Herman J. Achard, of Roselle, Ill.
Alphonso V. Bowker, of Athol.
James Burton, of Gilbertville.
Charles A. Davenport, of Newton.
Frank H. Drew.
Joseph E. Duxbury, of Boston.
George W. Greenleaf.
Perry E. Joslin, of Hopkinton.
Edward D. Laforce, of Central Falls, R. I.
Albert C. Leach, of Orange.
John C. McCarthy, of Malden.
Robert J. Mansfield of Springfield.
Jonathan H. Meigs, of Beachmont.
Frank O. Mosher.
Charles H. Quirk, of Buenos Ayres, S. A.
Edward O. Robinson.
Gilman P. Robinson, of Atlanta, Ga.
Alexander Rovinski, of Boston.
Albert A. Sargent, The Klondike.
Whitmell P. Small, of Great Barrington.
James S. Sullivan, of Randolph.

# Medical Communications

OF

# THE MASSACHUSETTS MEDICAL SOCIETY.

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# NOTICE TO FELLOWS OF THE SOCIETY.

1. The Society's MEDICAL COMMUNICATIONS are issued as soon as practicable after the Annual Meeting. The Publications are sent only to those who have paid their assessments, and to such Honorary and Retired Members as may annually apply for them.

2. The annual assessment should be paid in advance (*i.e.* before the close of the Annual Meeting), to the proper District Treasurer, or, in case of non-resident members, to the Treasurer of the Society, Dr. EDWARD M. BUCKINGHAM, 342 Marlborough Street, Boston. Payments thus made increase the annual dividend to the District Societies. Checks should be made payable to the order of The Massachusetts Medical Society.

3. Fellows who, having paid their assessments, fail to receive the Publications, should notify the Librarian, Dr. EDWIN H. BRIGHAM, 8, The Fenway, Boston.

4. Fellows wishing to become Retired Members, should observe the requirements of By-Law V., as follows:— "A Fellow who has arrived at the age of sixty-five years, has paid all his assessments, unless duly excused from them, may, upon a request in writing to the Treasurer of the Society, and on recommendation of the Committee on Membership and Finance and by a vote of the Councillors, become a Retired Member."

5. Fellows are requested to send promptly to the Librarian notice of changes of residence.

6. The next Annual Meeting of the Society will be held in Boston, on the second Wednesday in June, 1902. Meetings of Sections on the preceding day.

FRANCIS W. GOSS,
*Recording Secretary.*

217 *Warren Street, Roxbury, Mass.*